DRAFT

LOWER PASSAIC RIVER EARLY FINAL ACTION RISK EVALUATION

WAD 2, Work Element 3.5h

Submitted to

U.S. Environmental Protection Agency,
Region 2

and

U.S. Army Corps of Engineers,
Kansas City District

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Prepared by

Battelle
397 Washington Street
Duxbury, MA 02332
(781) 934-0571

Under Contract No. KC-ACE2002-18 to

Malcolm Pirnie, Inc.
104 Corporate Park Drive
White Plains, NY 10601-2100
914-694-2100

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ACRONYMS

ADD	Average daily dose
AE/WP	American eel and white perch
AT	Averaging time
ATSDR	Agency for Toxic Substances Disease Registry
BAF	Bioaccumulation factors
BERA	Baseline ecological risk assessment
BSAF	Biota-sediment accumulation factors
BW	Body weight
CalEPA	State of California Environmental Protection Agency
CARP	Contaminant Assessment and Reduction Program
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CL	Cooking loss
COPC	Contaminant of potential concern
COPEC	Contaminant of potential ecological concern
Cs	Cesium
CSF	Cancer slope factor
CSM	Conceptual site model
CSO	Combined sewer overflow
C_t	Biota tissue concentration
CTE	Central tendency exposure
DDD	Dichlorodiphenyldichloroethane
DDE	Dichlorodiphenyldichloroethylene
DDT	Dichlorodiphenyltrichloroethane
DDx	Sum of DDD, DDE, and DDT
D/F	Dioxin/furan
DSRT	NJ DEP's Division of Science, Research and Technology
DQO	Data quality objective
ED	Exposure duration
EF	Exposure frequency
EFA	Early Final Action
EFH	Exposure Factors Handbook
EPC	Exposure point concentration
ERE	Ecological risk evaluation
ESP	Ecological Sampling Program
FDA	Food and Drug Administration
FFS	Focused Feasibility Study
FI	Fraction ingested from contaminated source
g	Grams
HEAST	Health Effects Assessment Summary Tables
Hg	Mercury
HHRA	Human health risk assessment
HHRE	Human health risk evaluation
HI	Hazard index
HQ	Hazard quotient
IR	Ingestion rate
IRIS	Integrated Risk Information System
kg	Kilogram
LADD	Lifetime average daily dose
LOAEL	Lowest observed adverse effects level

LOED	Lowest observed effect dose
LPRRP	Lower Passaic River Restoration Project
MDL	Method detection limit
mg	Milligram
mg/kg-day	Milligram per kilogram of body weight per day
MRL	Minimal risk level
NA	Not applicable
NCP	National Oil and Hazardous Substances Pollution Contingency Plan
NJ	New Jersey
NJDEP	New Jersey Department of Environmental Protection
NOAEL	No observed adverse effects level
NPL	National Priorities List
NY	New York
NYSDEC	New York State Department of Environmental Conservation
NYSDOH	New York State Department of Health
PAH	Polycyclic aromatic hydrocarbon
PAR	Pathways Analysis Report
PCB	Polychlorinated biphenyl
PPRTVs	Provisional peer-reviewed toxicity values
ppb	Parts per billion (which is equivalent to microgram per kilogram or nanogram per gram)
ppm	Parts per million (which is equivalent to microgram per gram)
PREmis	Passaic River Estuary management information system
PRG	Preliminary remediation goal
QA/QC	Quality assurance/quality control
RAGS	Risk Assessment Guidance for Superfund
RfD	Reference dose
RI/FS	Remedial Investigation/Feasibility Study
RM	River mile
RME	Reasonable maximum exposure
SOP	Standard Operating Procedure
SOW	Statement of Work
STL	Severn-Trent Laboratories
SUF	Site use factor
SVOC	Semivolatile organic compound
TCDD	Tetrachlorodibenzo-p-dioxin
TEF	Toxic equivalency factor
TEQ	Toxic equivalence
TSI	Tierra Solutions, Inc.
TRV	Toxicity reference value
UCL	Upper confidence limit of the mean
ug/g	Microgram per gram (which is equivalent to parts per million)
USACE	Unites States Army Corps of Engineers
USEPA	United States Environmental Protection Agency
VOC	Volatile organic compound
WHO	World Health Organization

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1.0 INTRODUCTION

This document presents the human health and ecological risk evaluations to support the Early Final Action (EFA) Draft Focused Feasibility Study (FFS) for the Lower Passaic River Restoration Project (LPRRP). The Draft FFS evaluates alternative remedial actions within three target areas for the lower eight miles of the Lower Passaic River. These target areas have been developed based on sediment and bathymetric data collection and evaluation.

Human health and ecological risk assessments are designed to aid in risk management decisions regarding the actions necessary to address the hazardous substances at the site. In this document, risks associated with current conditions are estimated to assist United States Environmental Protection Agency (USEPA) in evaluating the appropriateness of undertaking remedial action(s). Risks under current conditions are then compared to future risks estimated to remain after remediation of specific target areas to support a detailed and comparative analysis of the various remedial alternatives and to support a decision by USEPA on selection of a remedial action. In addition to this assessment, a baseline human health risk assessment (HHRA) will be developed to support a final remedial decision for the Lower Passaic River.

As part of the Draft FFS, this document follows a screening level risk assessment approach based on USEPA (1989) Superfund risk assessment guidance. It provides the information necessary to develop a remedial action prior to the completion of a baseline risk assessment and a full Remedial Investigation/Feasibility Study (RI/FS). Additional refinements are anticipated to support a final remedial decision for the Lower Passaic River.

2.0 COMPILATION OF AVAILABLE DATA

The Lower Passaic River has been extensively sampled since the 1990s and those environmental sampling programs conducted since 1993 that were included in this analysis are summarized in Table 2-1. Analytical chemistry data derived from these studies were obtained from www.ourPassaic.org and the Contaminant Assessment and Reduction Program (CARP) and utilized to assess current and potential future cancer risks and noncancer health hazards to human and ecological receptors. Analytical data from fish and Blue crab tissue samples were used to estimate current cancer risks and noncancer health hazards, whereas analytical data from surface sediment samples were used to estimate future biota concentrations for use in evaluating potential future cancer risks and noncancer health hazards associated with the site following remedial action. Much of the analytical data were collected in the 1990s and may not be representative of current surface conditions in the river. Therefore, for this evaluation, only surface sediment and tissue data collected from 1993 to the present were used.

It should be noted that, based on the objectives of the risk evaluations and the schedule for implementing risk management decisions, the analytical data used in the human heath and ecological risk evaluations did not undergo a full data usability assessment following guidance from USEPA (1992). However, appropriate quality assurance/quality control (QA/QC) procedures appear to have been conducted on most datasets and the data are deemed to be of sufficient quality to perform these risk evaluations. It is anticipated that a complete evaluation of the usability of the risk assessment datasets will be conducted in preparation for the Baseline Human Health and Ecological Risk Assessments as part of the Lower Passaic River RI/FS. Table 2-1 provides a detailed list of the datasets that were utilized for this task and their QA/QC procedures, if available. The sampling locations for sediment are depicted in Figure 2-1 and biota sampling locations are depicted in Figure 2-2. A total of 12 sediment sampling locations, 10 from the 1994 sediment investigation and 2 from the 1999 Sediment Sampling Program, appeared outside the boundaries of the Passaic River (*i.e.*, on land) when plotted on a map using the sample coordinates available in the database. These samples were determined to be collected from the river so their locations were manually adjusted in Figures 2-1 and 2-2 to their approximate sample location in the river. The full dataset is provided in Attachment A.

Table 2-1. Summary of Data Used for the Risk Assessments.

Name of Study in Database	Depth (ft)	Number of Samples	River Mile Range	QA/QC Procedures[a]
Sediment				
PASSAIC 1994 Surficial Sediment Investigation[b]	0.5	40	3.5-6.9	Quantitative QA/QC[c]
PASSAIC 1995 USACE Minish Park Investigation	2	2	3.9-5.4	Not Specified
PASSAIC 1995 Sediment Grab Sampling Program	0.5	7	2.5-2.7	USEPA Region 2 Validation; full validation
PASSAIC 1995 RI Sampling Program	0.5	195	1.0-6.7	USEPA Region 2 Validation; full validation
PASSAIC 1997 Outfall Sampling Program	0.5	3	1.2-5.7	Quantitative QA/QC[c]
PASSAIC 1999 Sediment Sampling Program*	1.0	3	0.7-6.2	Quantitative QA/QC[c]
PASSAIC 1999 Late Summer/Early Fall ESP Sampling Program	0.5	48	1.0-6.9	USEPA Region 2 Validation
PASSAIC 1999/2000 Minish Park Monitoring Program	0.5	9	5.0-5.1	Quantitative QA/QC[c]
PASSAIC 2000 Spring ESP Sampling Program	0.5	17	1.0-6.8	USEPA Region 2 Validation
Pirnie Study (2005) HIGH RES CORE	0.6	44	1.4-3.5	USEPA Region 2 Validation or Third Party Full Data Validation[d]
Pirnie Study (2006) LOW RES CORE	2.3	31	2.9-6.7	USEPA Region 2 Validation or Third Party Full Data Validation[d]
NOAA NS&T Hudson-Raritan Phase II- 1993	0	1	7.37	Information not available
Pirnie Study Dredge Pilot Coring Program 2004 – Earth Tech	1.0	15	2.8-2.9	Third Party Full and Partial Data Validation[d]
Tissue				
NYSDEC 1993	NA	8	0.1	Information not available
PASSAIC 1995 Biological Sampling Program	NA	13	1.1-4.5	USEPA Region 2 Validation
PASSAIC 1999 Late Summer/Early Fall ESP Sampling Program	NA	267	1.0-6.9	USEPA Region 2 Validation
PASSAIC 2000 Spring ESP Sampling Program	NA	80	1.0-6.8	USEPA Region 2 Validation
PASSAIC 2001 RI Supplemental ESP Biota Sampling Program	NA	14	6.0-6.9	USEPA Region 2 Validation
CARP Datasets 2000-2004 Harbor Crustacean Collection Harbor Fish Collection	NA	67	2.6-10.0	Partial Third Party Validation[e]

[a.] QA/QC procedures from Passaic River Estuary management information system (PREmis) datasets as described by Tierra Solutions, Inc. (TSI) (2004).

[b.] The X and Y coordinates reported in the PREmis database for locations from these studies plotted on land. These points were relocated manually into the river in Figure 2-1 after conferring with Malcolm Pirnie and jointly concluding that these

were in fact sediment samples with incorrect position coordinates in the PREmis database. The 1994 Surficial Sediment Investigation points were moved due west until they were in the river. The 1999 Sediment Sampling Program points were moved together to the southeast until both points were just in the water, on opposite shores.

[c.] Quantitative QA/QC includes the analysis of field and laboratory duplicates, rinsate blanks, matrix spike/matrix spike duplicates, and other quantitative measures of precision and accuracy but without specification of implementing USEPA Region 2 data validation procedures.

[d.] Data validation activities were performed by Severn-Trent Laboratories (STL) in accordance with the USEPA Method, the Laboratories Standard Operating Procedure (SOP) and the Malcolm Pirnie, Inc. Statement of Work (SOW).

[e.] In addition to internal QA/QC procedures, partial datasets were verified by Booz Allen Hamilton.

NA = Not Applicable
USACE = United States Army Corps of Engineers
RI = Remedial Investigation
ESP = Ecological Sampling Program
CARP = Contaminant Assessment and Reduction Program

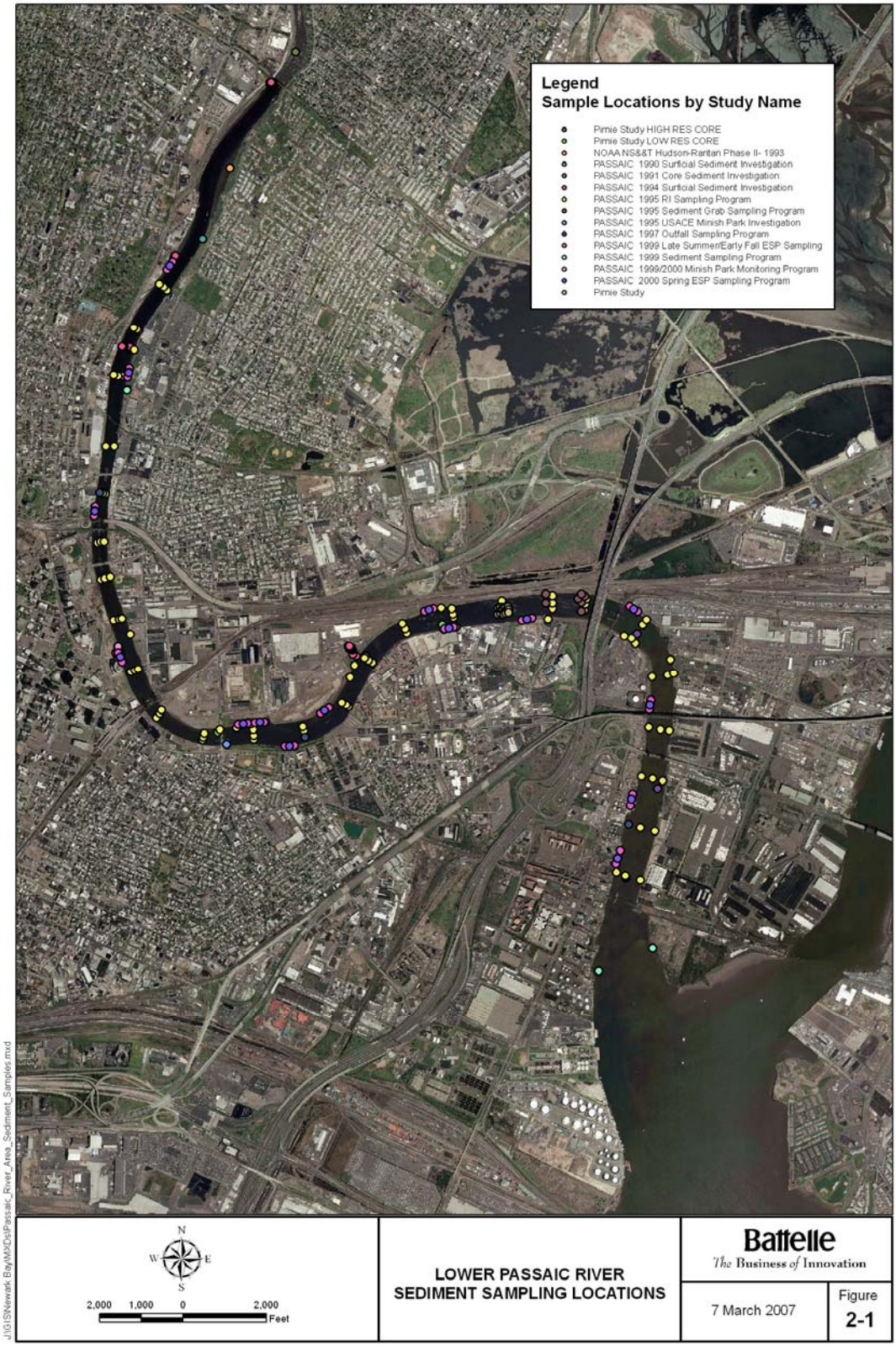


Figure 2-1. Sediment Sampling Locations along the Lower Eight Miles of the Passaic River.

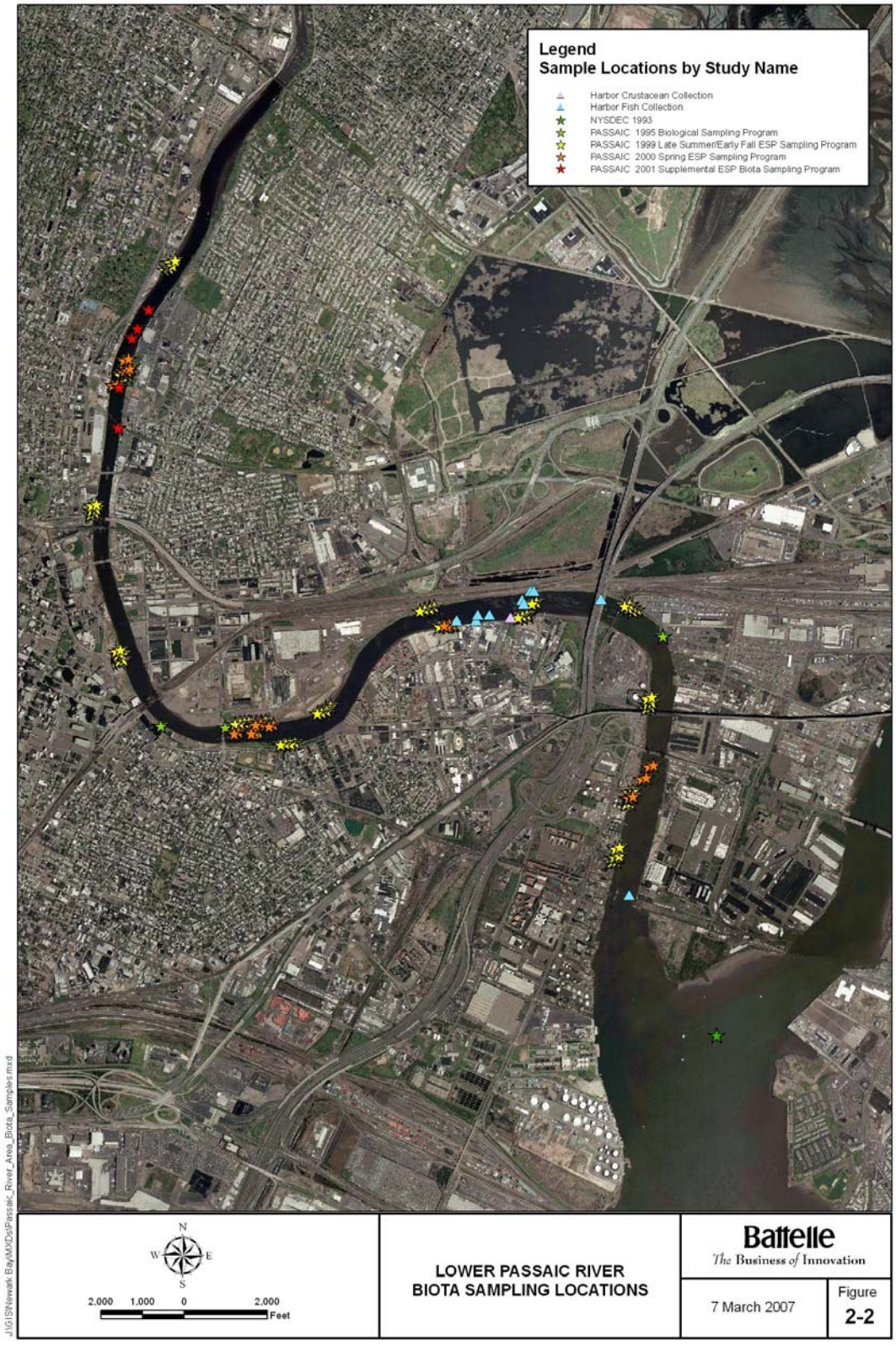


Figure 2-2. Biota Sampling Locations along the Lower Eight Miles of the Passaic River.

In this biological and sediment data review the following chemical classes were examined:

- Polychlorinated dibenzodioxins (dioxins);
- Polychlorinated dibenzofurans (furans);
- Polychlorinated biphenyl (PCB) congeners;
- PCB Aroclors;
- Pesticides;
- Polycyclic aromatic hydrocarbon (PAH); and
- Metals.

In addition, the following Toxic Equivalency (TEQ) calculations were performed for dioxins/furans and coplanar (dioxin-like) PCB congeners:

- TCDD TEQ for dioxin/furans – sum the products of the congener concentration and congener-specific Toxic Equivalency Factors[1] (TEF) for all dioxin/furan congeners (Table 2-2);
- TCDD TEQ for PCB Coplanar Congeners – sum products of the congener concentration and their TEFs (Table 2-2) for 12 coplanar PCB compounds [i.e., the World Health Organization (WHO) congeners]; and
- Total TCDD TEQ – the sum of the above two results.

The tissue data were obtained from the database prepared for the Geochemical Evaluation Biota Plots (Malcolm Pirnie, 2006). Non-detects (data qualifiers including "U" or as "ND") were reported as one-half the detection limit value for all database exports including sums and individual parameters. New queries were added to calculate the TEQ values for dioxin/furans, PCB congeners, and total chlordane. Table 2-3 provides a summary of the matrices sampled for crab and fish tissue. For crab, all matrices comprised the dataset for the human health and ecological risk evaluations. For fish, all matrices listed in Table 2-3 were used to compile the data for both the human health and ecological evaluations. Information regarding the specific characteristics of crab and fish samples was missing from the database. This includes data that would correlate with the age, length, weight, and sex of the fish. This lack of information could be a potential cause of uncertainty in the estimation of exposure concentrations and whether the fish/crab was of a consumable size for human consumption.

Sediment data for the various studies were loaded into a Microsoft Access database and the data were compiled into one table. All queries and calculations use one-half the detection limit where qualifiers are reported as "U" or "ND". It is recognized that this may overestimate Aroclor totals and this is discussed further in the uncertainty section.

Data queries were performed using Microsoft Access for each parameter group of interest. The individual analytical results were summed in Access for the following chemicals: Total DDx, (sum of dichlorodiphenyldichloroethane (DDD), dichlorodiphenyldichloroethylene (DDE), and dichlorodiphenyltrichloroethane (DDT), and PCB Aroclors. It was noted that the data extracted from the project database did not always include all parameters used for the summation (*e.g.*, there were not always data for all seven PCB Aroclors). The reason for these inconsistencies was not investigated and the summations were calculated based only on the available parameters.

[1] A TEF is a measure of the relative potency of a compound to cause a particular toxic or biological effect relative to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). By convention, TCDD is assigned a TEF of 1.0 and the TEFs for other compounds with dioxin-like effects ranging from 0 to 1. When TEFs are derived based on the relative binding affinity to the aryl hydrocarbon (Ah) receptor or induction of cytochrome P4501A1, it is assumed that these biochemical responses correlate with toxicologically important effects (Van den Berg *et al*., 1998).

TEQ values for dioxin/furans and PCB congeners were calculated using congener-specific TEFs (Table 2-2). The TEFs were calculated separately for each individual parameter and summed to derive the TEQ value.

Table 2-2. Toxic Equivalency Factors for Dioxin/Furans and Dioxin-like PCB Congeners.

Congener	1998 Mammal TEF	2005 Mammal TEF	Fish TEF	Bird TEF
Dioxins/Furans				
2,3,7,8- TCDD	1	1	1	1
1,2,3,7,8-PeCDD	1	1	1	1
1,2,3,4,7,8-HxCDD	0.1	0.1	0.5	0.05
1,2,3,6,7,8-HxCDD	0.1	0.1	0.01	0.01
1,2,3,7,8,9-HxCDD	0.1	0.1	0.01	0.1
1,2,3,4,6,7,8-HpCDD	0.01	0.01	0.001	0.001
OCDD	0.0001	0.0003	0.0001	0.0001
2,3,7,8-TCDF	0.1	0.1	0.05	1
1,2,3,7,8-PeCDF	0.05	0.03	0.05	0.1
2,3,4,7,8-PeCDF	0.5	0.3	0.5	1
1,2,3,4,7,8-HxCDF	0.1	0.1	0.1	0.1
1,2,3,6,7,8-HxCDF	0.1	0.1	0.1	0.1
1,2,3,7,8,9-HxCDF	0.1	0.1	0.1	0.1
2,3,4,6,7,8-HxCDF	0.1	0.1	0.1	0.1
1,2,3,4,6,7,8-HpCDF	0.01	0.01	0.01	0.01
1,2,3,4,7,8,9-HpCDF	0.01	0.01	0.01	0.01
OCDF	0.0001	0.0003	0.0001	0.0001
PCB Congeners				
3,3',4,4'-Tetrachlorobiphenyl (77)	0.0001	0.0001	0.0001	0.05
3,4,4',5-Tetrachlorobiphenyl (81)	0.0001	0.0003	0.0005	0.1
3,3',4,4',5-Pentachlorobiphenyl (126)	0.1	0.1	0.005	0.1
3,3',4,4',5,5'-Hexachlorobiphenyl (169)	0.01	0.03	0.00005	0.001
2,3,3',4,4'-Pentachlorobiphenyl (105)	0.0001	0.00003	0.000005	0.0001
2,3,4,4',5-Pentachlorobiphenyl (114)	0.0005	0.00003	0.000005	0.0001
2,3',4,4',5-Pentachlorobiphenyl (118)	0.0001	0.00003	0.000005	0.00001
2',3,4,4',5-Pentachlorobiphenyl	0.0001	0.00003	0.000005	0.00001
2,3,3',4,4',5-Hexachlorobiphenyl (156)	0.0005	0.00003	0.000005	0.0001
2,3,3',4,4',5'-Hexachlorobiphenyl	0.0005	0.00003	0.000005	0.0001
2,3',4,4',5,5'-Hexachlorobiphenyl (167)	0.00001	0.00003	0.000005	0.00001
2,3,3',4,4',5,5'-Heptachlorobiphenyl (189)	0.0001	0.00003	0.000005	0.00001

Source: Van den Berg *et al.,* 1998; Van den Berg *et al.,* 2005.

Table 2-3. Sample Matrices for Crab and Fish Tissue.

Sample Type	Matrix
Crab	All edible tissue
	Hepatopancreas
	Muscle
	Tissue
Fish	Whole organism
	Tissue
	Whole organism without the head and viscera

3.0 IDENTIFICATION OF CONTAMINANTS OF POTENTIAL CONCERN (COPC)

Conclusions presented in the Pathways Analysis Report (PAR) (Battelle, 2006a) identified several classes of contaminants of potential concern (COPC), including various metals, pesticides, PAHs, dioxins/furans, PCBs, and volatile and semivolatile organic compounds (VOC/SVOC). For human health, no additional screening for COPCs was performed to support the Draft FFS, but rather, a subset of the COPCs identified in the PAR was used to capture the primary risk drivers and carried through the risk assessment process. For the ecological evaluation, a more refined screening analysis of COPCs was conducted for the Draft FFS. This technical memorandum documents the screening process used to develop a refined list of ecological COPCs and is presented in Attachment D.

COPCs for the Human Health Evaluation

For human health, COPCs evaluated in the Draft FFS represent those compounds that are considered most bioaccumulative, most persistent in the environment, and are relatively toxic to human and ecological receptors. In addition, these COPCs represent the contaminants that have triggered states to issue fish and shellfish consumption advisories or bans (USEPA, 2000a; USEPA, 2005a). USEPA (2005a) reports that there are advisories in the United States for 36 chemical contaminants; however 98% of these advisories in effect in 2004 involved five bioaccumulative chemicals, including mercury, PCBs, chlordane, dioxins, and DDT. The larger set of COPCs identified in the PAR will be assessed as part of the RI/FS process. Human health COPCs identified for this evaluation are summarized on Table 3-1 and include the following:

- Dioxins/furans (as TCDD TEQ);
- Total PCBs (sum Aroclors);
- PCBs (12 dioxin-like congeners as TCDD TEQ);
- DDE, DDD, and DDT;
- Dieldrin;
- Total chlordane; and
- Mercury (including methyl mercury).

Data for total mercury and methyl mercury were assumed to be equivalent and treated as if all were methyl mercury. Once mercury is released to the environment it can be converted to a biologically toxic

form of methyl mercury. Methyl mercury is of particular concern because it readily crosses biological membranes and can accumulate and biomagnify up the food chain (Brightbill *et al*., 2004). Most of the mercury consumed in fish or other seafood is the highly absorbable methyl mercury form (Agency for Toxic Substances and Disease Registry [ATSDR], 1999). USEPA (2000a) recognizes that most mercury in fish and shellfish tissue is present as methyl mercury, but because of the relatively high analytical cost for methyl mercury, recommends total mercury be determined and then conservatively assume all of the mercury present is methyl mercury. Therefore, due to lack of methyl mercury analytical results in the tissue dataset used for this HHRE, analytical results for mercury were used as a surrogate for methyl mercury, which assumes that all the mercury detected in the tissue is methyl mercury. Various studies, as summarized in USEPA (2000a), report that mercury concentrations are greater in higher trophic level fish species. Studies conducted to assess the correlation between total mercury and methyl mercury in fish tissue (Grieb *et al*., 1990; Bloom, 1992; and Kannan *et al*., 1998) reported contributions of methyl mercury to total mercury ranged between 83% up to more than 99%. Most of the data available for this risk assessment consisted of elemental mercury, which were therefore assumed to be methyl mercury. As a result of this assumption, exposure point concentrations (EPC) derived using mercury data may slightly overestimate the methyl mercury concentration.

PAHs were not selected as COPCs for human health. Although potentially toxic to certain fish species, PAHs are not expected to bioaccumulate in the tissues of aquatic organisms because fish and most crustaceans have the ability to metabolize PAHs and eliminate the breakdown products in feces and urine (ATSDR, 1995).

COPECs for Ecological Evaluation

For the ecological risk evaluation in support the FFS, sediment contaminants of potential ecological concern (COPEC) were identified based on a three-tier screening process that included the following factors:

1. Bioaccumulation screen (indirect toxicological effects to wildlife through the food chain);
2. Essential nutrient screen; and
3. Effects value screen (direct toxicological effects to benthic invertebrates).

The screening process is described in detail in Attachment D. Ten COPECs (Table 3-1) were identified as comprising the largest contribution of total potential risk and were carried through this evaluation. These compounds had hazard quotients (HQs) that exceeded 100 for inorganic compounds and greater than 1,000 for organic compounds. Ecological COPECs identified for this evaluation include the following:

- Dioxins/furans (as TCDD TEQ);
- PCB congeners (12 dioxin-like congeners as TCDD TEQ);
- Total PCB (sum Aroclors);
- Total DDx (sum of DDE, DDD, and DDT isomers);
- Dieldrin;
- Low molecular weight PAHs;
- High molecular weight PAHs;
- Copper;
- Lead; and
- Mercury (including methyl mercury).

As done in the human health assessment, data for total mercury and methyl mercury were assumed to be equivalent and treated as if all were methyl mercury.

Table 3-1. Summary of Contaminants of Potential Concern for Human Health and Contaminants of Potential Ecological Concern.

Analyte	COPC for Human Health Assessment	COPEC for Ecological Assessment
Inorganic Compounds		
Copper		√
Lead		√
Mercury	√	√
Semivolatile Organic Compounds (PAHs)		
Low molecular weight PAHs		√
High molecular weight PAHs		√
Polychlorinated biphenyl (PCB)		
Total PCBs (sum Aroclors)	√	√
TCDD TEQ (PCBs)	√	√
Pesticides/Herbicides		
Chlordane	√	
Dieldrin	√	√
DDE	√	
DDD	√	
DDT	√	
Total DDx		√
Dioxin and Furans		
TCDD TEQ (D/F)	√	√

4.0 HUMAN HEALTH AND ECOLOGICAL CONCEPTUAL SITE MODELS

An overall project conceptual site model (CSM) is a multidisciplinary tool that serves a critical role in risk assessment, numerical modeling development, project, and sample planning, decision making, and ultimately in developing a remedial strategy. The CSM is developed during the first step of the data quality objective (DQO) process (USEPA, 2006) and continues to evolve throughout the project as historical and recently collected data are evaluated, DQOs are updated, and risk assessments are refined. Typical risk assessment components of a CSM include the following:

- Potential source of contamination;
- Potentially contaminated media and types of contaminants;
- Contaminant fate and transport mechanisms and migration pathways;

- Potential exposure pathways; and
- Potential human and ecological receptors.

The risk assessment CSM for the Lower Passaic River includes the lower 17 miles of the river, from the Dundee Dam to the confluence with Newark Bay (see Appendix A of the Draft FFS). The river has been divided into three sections, based on salinity measurements and geomorphology. The freshwater section, with salinity values less than 0.5 parts per thousand (‰) extends from the Dundee Dam to river mile (RM) 9. The transitional section represents the portion between the freshwater and brackish sections, where the salt wedge typically advances under high-tide conditions. Here, water conditions can range from slightly brackish (0.5 to 5.0 ‰) to moderately brackish (5.0 to 18 ‰). The brackish section has almost always moderately brackish conditions, with salinities ranging from 5 to 18 ‰. This Draft FFS focuses on the lower and brackish sections of the river, extending up to RM 8. Individual CSMs were developed for the human health and ecological risk evaluations to define the exposure pathways for each assessment.

4.1 Nature and Extent of COPCs

The Lower Passaic River has been used as a major means of conveyance for municipal discharges from the middle of the 19th century to the present time. Together, these waste streams (industrial and municipal) have delivered a number of contaminants, including 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD), PAHs, PCBs, DDT, mercury, lead, and others. There have been major physical changes to the river over this period as well. Several large dredging projects were undertaken at the beginning of the twentieth century to create a ship channel to RM 15. However, since the 1940s, there has been little maintenance dredging above RM 2. Consequently, the channel has extensively filled back in, particularly between RM 2 and RM 8. The coincidence of chemical disposal in the river along with the construction, and subsequent limited maintenance, of the navigation channel created an ideal situation for the accumulation of contaminated sediments. As a result, the river has accumulated substantial sediment beds, measuring 15 feet thick or more in some areas. These thick beds exist primarily below RM 8, where the wider river channel has permitted rapid sediment accumulation, as compared to the narrower channel conditions farther upstream. Relatively little accumulation has occurred upstream of this point.

Despite the prevalence of thick sediment deposits below RM 8, the sediments in this region are not all stable, and erosional areas can be found throughout the lower eight miles of the river. Some or all of these erosional areas are believed to be responsible for on-going release of contaminants from the river bed. A detailed examination of sediment deposition rates between RM 0.9 and RM 7 indicates a high degree of spatial heterogeneity, with local rates varying from about -6 inches/year of erosion to about +8 inches/year of deposition. Historical deposition rates were probably higher than current rates because of the more extensive salt intrusion present immediately after the initial channel dredging, which enhanced trapping of suspended matter. Based on solids balance considerations, current head-of-tide solids load to the Lower Passaic River is greater than the annual average rate of accumulation in the river. However, the historical rates of sediment accumulation in the Lower Passaic River were probably too large to be sustained solely by the Passaic's head-of-tide solids loads, suggesting that a net solids transport from Newark Bay supplied the additional solids.

The chemical contamination associated with the Lower Passaic River is largely driven by the contaminant burdens contained within the sediments, particularly for 2,3,7,8-TCDD. While on-going external inputs may exist, the concentrations within the sediments are responsible for much of the contamination within the water column. In fact, the legacy of contamination in the sediments probably extends back at least to the mid-nineteenth century. The oldest contaminants found in the sediments are PAH compounds, cadmium, mercury, and lead, which probably pre-date the turn of the 20th century. Following these contaminants are, in order of appearance in the river, DDT, 2,3,7,8-TCDD, and PCBs. Other

contaminants, such as arsenic, chromium, and copper, are also present in the sediment record. The available evidence indicates that several of these compounds (*i.e.,* PAHs, PCBs, mercury, and lead) at least partially originated above the head-of-tide and Dundee Dam. Others, like 2,3,7,8-TCDD and DDT, are nearly exclusively the result of discharges to the Lower Passaic River.

One important observation from the extent of chemical contamination in the Lower Passaic River is extent of tidal mixing throughout the river. Recently deposited sediments anywhere within the Lower Passaic River have very similar concentrations of contaminants, indicating that sediments are well homogenized prior to deposition. Thus, the presence or absence of an interval of high concentration within the sediments at a given location is a function of the depositional history at that location and is generally not controlled by proximity to source. As a result, thick sequences of contaminated sediments will tend to have similar inventories of contaminants regardless of their location in the river. The coring data that form the basis for estimating these inventories show a high degree of local spatial heterogeneity, indicating that localized areas of relatively higher concentrations typically described as "hot spots" do not exist. Instead, "hot" regions of the river typically exist on the scale of a mile or more, nearly bank to bank in lateral extent. This understanding underlies the delineation of remedial target areas used as a basis to develop remedial alternatives.

The three target areas for remediation were identified using geochemical evaluations, analytical results from the low resolution cores, and evaluations of the bathymetric data. As an outcome of these analyses, candidate target areas for remediation are identified as follows:

- **Primary Erosional Zone:** Locations adjacent to erosional zones between RM 3.45 and RM 5.05;
- **Primary Inventory Zone:** Locations that are consistently depositional with high contaminant inventory between RM 2.4 and RM 3.3; and,
- **Area of Focus:** The entire bank-to-bank river area from RM 0 to RM 8, including both erosional and depositional areas.

4.2 Exposure Assessment

The objective of the exposure assessment is to estimate the magnitude, frequency, duration, and routes of current and reasonably anticipated future exposure to COPCs associated with the site. The exposure assessment is based on the receptor scenarios described in the conceptual site models that define the conditions of exposure to site-related COPCs.

An exposure pathway defines the most probable pathway in which a receptor may come in contact with a contaminated medium. For an exposure pathway to be complete, the following four elements must be present:

1. A source and mechanism of chemical release;
2. A retention or transport medium;
3. A point of contact between the receptor and the medium; and
4. A route of exposure for the potential receptor at the contact point.

There must be a complete exposure pathway from the source of chemicals in the environment (*i.e.,* from sediment or biota tissue) to receptors for chemical intake to occur. If at least one exposure pathways is complete, chemical intake may occur and adverse effects may be associated with site-related COPCs.

The complete exposure pathways identified in the PAR (Battelle, 2006a) are:

- Direct contact with surface water and/or sediment;

- Inhalation, incidental ingestion of sediment and/or surface water; and
- Ingestion of fish/shellfish.

A summary of each of the relevant pathways with respect to human and ecological health are provided in the following sections.

4.2.1 Human Exposures

Currently, the banks of the Lower Passaic River are extensively developed and surrounded by a mixture of residential, commercial, and industrial activities. Intensive commercial and industrial uses occur in the area due to a highly developed transportation infrastructure that includes highway, railway, and marine services. Individuals are known to catch fish and crab along the river banks and from docks and bulkheads (May and Burger, 1996; Burger *et al*., 1999; Kirk-Pflugh *et al.,* 1999). In addition, there are several rowing clubs that engage in crew and other boating activities for adults and children. Furthermore, there are a few parks, docks, and mudflat areas that currently are used by residents and visitors for recreational purposes. Based on this information and ongoing initiatives to restore the Passaic River, it was assumed that exposure to contaminants in the river would be associated with current recreational activities such as swimming, wading, fishing, crabbing, and boating. Human receptors identified as engaging in these activities include a Recreational User and an Angler/Sportsman. In addition, a transient community has occasionally constructed temporary housing along the banks of the river. There is limited information regarding the length of their occupancy and their activities while on the river, however, a residential scenario (homeless resident) was also included in the CSM to address potential exposures to this community. The receptors and exposure scenarios associated with future use are not expected to differ significantly from those being evaluated under the current use scenarios. A summary of each of these receptors and the complete exposure pathways associated with each is provided below and depicted on Figure 4-1.

Angler/Sportsman: The angler/sportsman is defined as an adult individual catching and consuming a variety of fish (*i.e*., carp, striped bass, catfish, and American eel), and other local species (*i.e*., Blue crab) from the river and surrounding areas. In addition, the possibility that individuals might also catch and consume other species such as waterfowl, turtles, or frogs from the river will be considered. The collection and consumption of fish and shellfish from the Passaic River has been well documented (Belton *et al*., 1985; May and Burger, 1996; New Jersey Department of Environmental Protection [NJDEP], 2002); therefore, it is clear that this exposure pathway is complete for the angler/sportsman. Consumption of other species is more speculative at this time and additional information will be required to evaluate this pathway quantitatively since there are no historical data on chemical concentrations in the tissues of these organisms. It is assumed that an adult angler/sportsman shares his/her catch with an adolescent (age 10-18 years) and a child (age 0-6 years) family member. Evaluation of subsistence fishing is not proposed in this assessment because there is no evidence that there are any individuals that rely solely on his/her daily catch. Direct exposures (*i.e.*, dermal contact and incidental ingestion) to sediments and surface water contacted during collection activities are potential pathways relevant to the adult/sportsman. Inhalation exposures may also occur if activities occur in areas where VOCs are present in sediments or surface water. It is assumed that any children accompanying the angler/sportsman during these activities would engage in typical recreational activities defined under the Recreational User scenario.

Recreational User: Recreational use along the Passaic River includes swimming, wading, and sculling. Because the likelihood of swimming in the Passaic River depends on the location along the river, it may not be appropriate to include swimming as a potential means of exposure at all locations. When swimming is feasible, exposure to chemicals in surface water and sediment are likely. Wading includes an individual walking around the mudflat areas, as wells as along shallower parts of the river; thus,

exposure is primarily to sediment, but may include exposure to surface water as well, depending on the location on the river. Scullers, for the most part, are expected to remain in their boats except for the occasional fall into the river where exposure to surface water and sediment is likely. For swimming and wading recreational activities, an adult, a child (age 0-6 years), and an adolescent (age 10-18 years) are all potential receptors. The sculler can be an adult or an adolescent. Potential exposure pathways identified are direct contact (ingestion and dermal contact) with sediment and surface water and inhalation exposures if activities occur in mudflat areas or near sediment where VOCs are present. Ingestion of fish and other biota has been identified only for the angler because his/her exposure would be higher than that of the recreational user.

Homeless Resident: Observations have been made that a number of transient individuals live in temporary makeshift shelters along the banks of the Passaic River. Although minimal information is available regarding the daily routine of these individuals, it is assumed that they would likely contact sediment and surface water during daily activities. Therefore, the Homeless Resident scenario evaluates the potential risks to an adult, a child (age 0-6 years), and an adolescent (age 10-18 years) living along the river. The adult and child exposures will be evaluated separately since it is unlikely that a child introduced to this lifestyle would continue to reside near the Passaic River into adulthood. Complete exposure pathways associated with this receptor are direct contact (ingestion and dermal contact) with sediment and surface water, ingestion of fish/other biota, and inhalation exposures if activities occur in mudflat areas or near sediment based on the presence of VOCs in the environment.

Based on the results of other Superfund HHRAs conducted for similar river sites and COPCs having the potential to bioaccumulate such as dioxins and PCBs (*e.g.*, Hudson River [TAMS Consultants, Inc. and Gradient Corporation, 2000]; Housatonic River [Weston Solutions, 2005]; Centredale Manor Woonasquatucket River [USEPA Region 1, 2005]), consumption of fish and shellfish is anticipated to be associated with the highest cancer risks and non-cancer health hazards compared to ingestion, dermal contact, or inhalation of chemicals in surface water or sediment. Despite New Jersey's fish/crab consumption advisories and prohibitions on taking or attempting to take Blue crabs in the Newark Bay Complex, NJDEP determined through angler surveys that fishing and crabbing continue to occur in this area (NJDEP, 1995; Kirk-Pflugh, *et al*., 1999). Thus, NJDEP used the consumption pattern data obtained from the angler surveys and dioxin concentration data for crabs collected from three studies of the Newark Bay Complex to estimate a range of cancer risks for consumption of regionally caught crabs (NJDEP, 2002). The NJDEP estimated the lifetime excess risk from consumption of crabs from the Newark Bay Complex ranged from 5,000 per million (0.005) to more than 1 million per million (NJDEP, 2002).

Therefore, for the purposes of this Draft FFS, exposures to an adult angler/sportsman and other family members (*i.e.*, adolescent and child) are the only receptors evaluated for exposure to COPCs associated with consumption of self-caught fish and Blue crab. Carcinogenic risks and noncarcinogenic health hazards will be estimated using exposure assumptions provided in the PAR (Battelle, 2006a) specifically for the angler/sportsman. Recreational swimming, wading, and boating are also complete exposure pathways that will be evaluated as part of the final RI/FS for the entire 17 miles of the Lower Passaic River.

4.2.2 Ecological Exposure

A wide range of ecological receptors is potentially at risk from COPECs in the Lower Passaic River, including benthic invertebrates, fish, and a variety of piscivorous or aquatic avian and mammalian predator species.

To estimate current and future risk to ecological receptors in the lower eight miles, benthic invertebrates and two upper-trophic level piscivorous receptors, the great blue heron and mink, were selected to represent bird and mammal populations, respectively. These species were selected as conservative surrogates because great blue heron are particularly sensitive to pesticides and mink are particularly sensitive to dioxin and PCBs. Mummichogs were selected as a conservative surrogate to represent the demersal forage fish. They are relatively common in the area and provide a forage food base for the upper-trophic level wildlife species. In addition, risk to piscivorous fish (*i.e.,* predatory fish that consume smaller fish) was also evaluated using data on American eel and white perch (AE/WP). The current ecological CSM for the Lower Passaic River is presented in Figure 4-2.

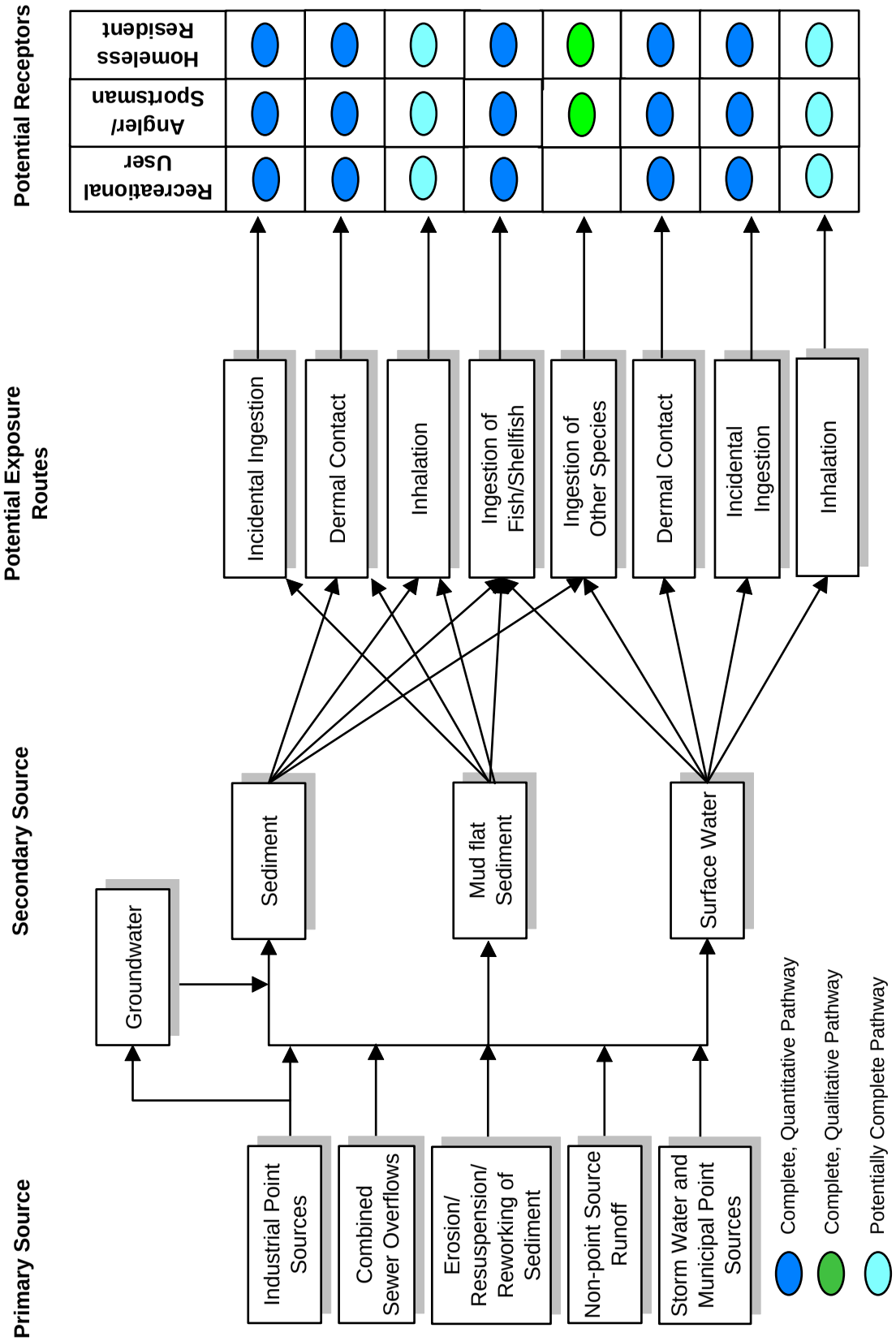


Figure 4-1. Human Health Conceptual Site Model.

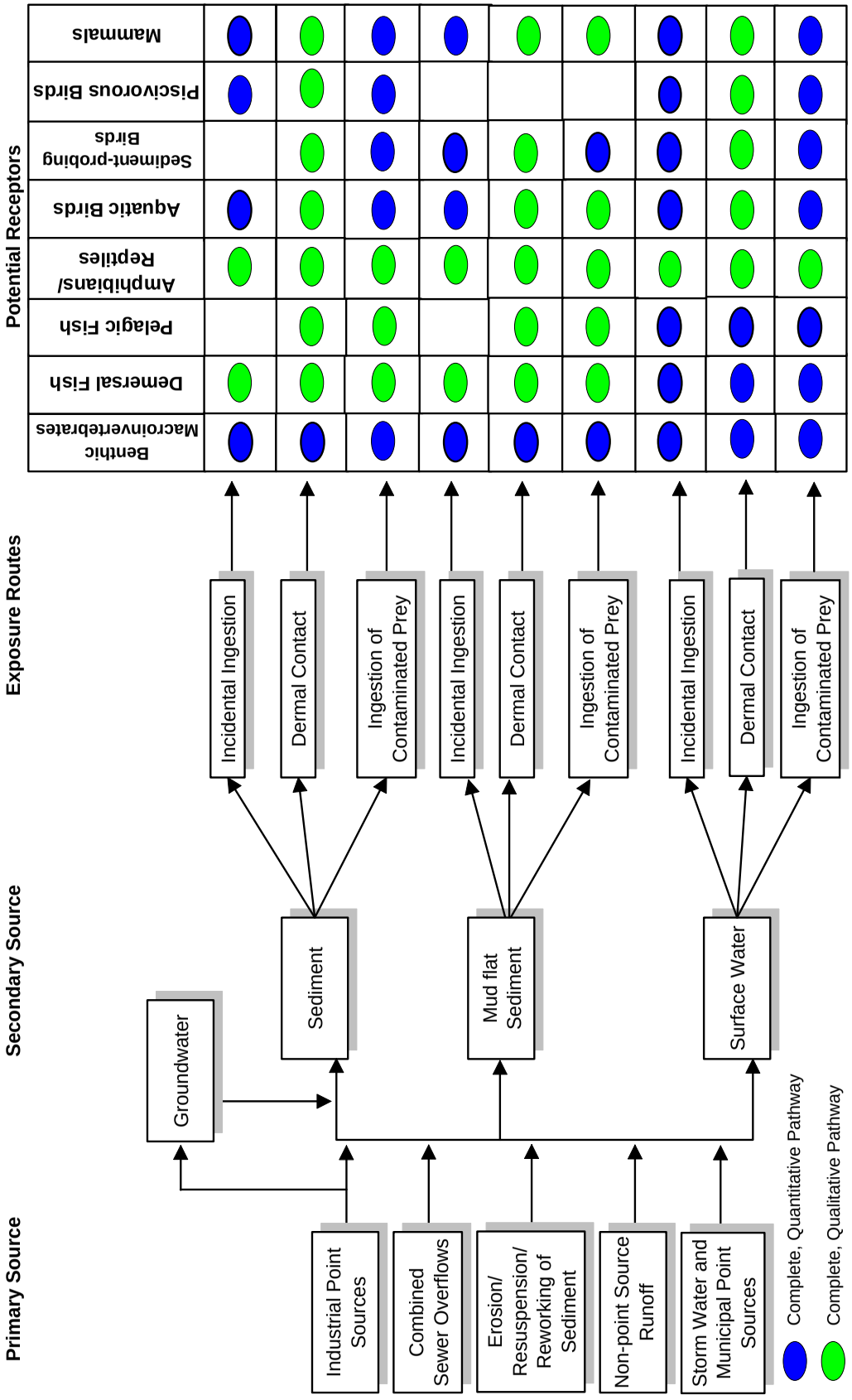


Figure 4-2. Ecological Conceptual Site Model.

4.2.3 EPC Development for Comparative Risk Evaluation

Estimates of chemical concentrations at points of potential exposure are necessary for evaluating chemical intakes by potentially exposed receptors. The concentrations of chemicals in the exposure medium at the exposure point are termed "exposure point concentrations" (EPC). USEPA guidance uses an average concentration to represent "a reasonable estimate of the concentration likely to be contacted over time" (USEPA, 1989) and "because of the uncertainty associated with estimating the true average concentration at a site" recommends that the 95 percent upper confidence limit (UCL) on the average be used.

Calculation of the EPCs followed guidance provided by USEPA (2002a), using distribution shift tests to determine the underlying population distribution. Specifically, the ProUCL software package (version 3.0) developed by USEPA (2004) was used to determine the underlying distributions and to determine the most applicable EPC for a given contaminant based on the characteristics of the data. Depending on the statistical distributions identified by the software application, the program provides a recommended EPC. For those cases when more than one estimate of the UCL is recommended by the software program, the first value is chosen as the UCL. When evaluating data, one-half the detection limit (USEPA, 1989) was used to represent non-detected values. The output files for each of the COPCs for human and ecological receptors from EPA ProUCL software are provided in Attachment B. A summary of the EPCs for sediment and tissue[2] is provided in Table 4-1.

[2] EPCs for tissue samples were based on direct measures of concentrations in biota and were not derived from models which predict tissue concentrations from sediment. As such, it was not necessary to lipid-normalize chemical concentrations for this evaluation.

Table 4-1. EPCs Based on 95% UCLs on the Arithmetic Mean for Sediment and Tissue.

COPC		95% UCLs[a] (µg/g = ppm)				
		Sediment	**Eel/ Perch[b]**	**Mummichogs[c]**	**Crab[d]**	**Crab[e]**
Chlordane		0.041	1.8	0.04	0.037	0.037
Copper[f]		236	25	3.9	NA	35
Dieldrin		0.019	0.027	0.0042	0.018[g]	0.022
Lead[f]		375	0.63	1.2	NA	0.55
Mercury		3.6	0.35	0.041[h]	0.097	0.097
LPAH[f]		41	0.17	0.17	NA	0.15
HPAH[f]		61	0.1	0.065	NA	0.16
TCDD TEQ (D/F)	Mammal	0.0016	0.00025	0.00014	0.00022	0.00022
	Bird	0.0018	0.00028	0.00015	NA	0.00027
	Fish	0.0016	0.00025	0.00014	NA	0.00022
TCDD TEQ (PCB)	Mammal	0.000045	0.000076	0.000027	0.0004	0.00044
	Bird	0.00075	0.00086	0.0002	NA	0.0028
	Fish	0.0000038	0.0000051	0.0000017	NA	0.000025
TCDD TEQ (D/F/PCB)	Mammal	0.0017	0.00022	0.00017	NA	0.00042
	Bird	0.0024	0.0011	0.00031	NA	0.0038
	Fish	0.0016	0.00026	0.00015	NA	0.00047
Total PCBs[i]		1.8	3.4	0.72	5.18	5.5
DDD		0.214	0.15	NA	0.138	NA
DDE		0.094	0.303	NA	0.317	NA
DDT		0.096	0.076	NA	0.235	NA
Total DDx		0.38	0.519	0.088	NA	0.56[j]

a. UCLs calculated based on the data queries from PREmis and CARP databases; samples included in the UCL calculations are listed in Attachment A. 95% UCLs on the mean calculated using USEPA ProUCL software (version 3.0); output files are included in Attachment B.

b. EPC derived from a combination of AE/WP tissue concentrations.

c. EPC derived from tissue concentration of mummichog for ecological evaluation.

d. EPC derived from edible Blue crab tissue data (muscle only) for human health evaluation.

e. EPC derived from entire Blue crab data (including hepatopancreas) for ecological evaluation.

f. Low Molecular Weight PAH (LPAH) and High Molecular Weight PAH (HPAH) added as ecological COPECs to the list evaluated in the Pre-Draft EFA FFS analysis (Battelle, 2006), based on a screening benchmark analysis (see Attachment D).

g. EPC based on maximum concentration rather than 95%UCL value.

h. In the instance when ProUCL recommended more than one value, the first value (Student's-t UCL) was selected.

i. Total PCBs represent the non-dioxin-like PCBs

j. The EPC for total DDx is less than the sum of the EPCs for DDD, DDE, and DDT as a result of calculating 95% UCLs.

NA = not applicable
D/F = dioxin/furan
µg/g = microgram per gram which is equivalent to ppm = parts per million

5.0 HUMAN HEALTH RISK EVALUATION – CURRENT CONDITIONS

This section describes the methodology and results of the current human health risk evaluation (HHRE) based on potential exposure of human receptors to COPCs in fish and crab tissue as identified in Section 4.0. The HHRE was conducted according to USEPA's RAGS Volume I, Human Health Evaluation Manual (Part A) (USEPA, 1989), and other appropriate USEPA guidance, guidelines and policies, including RAGS Part D (USEPA, 2001).

The purpose of the HHRE is to assess and document the magnitude of potential risk to human receptors based on current exposure to COPCs within the Lower Passaic River (RM 0 to RM 8), in the absence of remedial action. In addition, the risk evaluation provides an assessment of the overall cancer risks and non-cancer hazards to human health considering a "no action" approach, which serves as a basis for comparison for the remediation of contaminated sediment options proposed for the three target areas to address requirements in National Oil and Hazardous Substances Pollution Contingency Plan (NCP) Section 300.430(e)(9)(iii). The results of the evaluation will be used to inform risk management decisions regarding the potential remedial action.

5.1 Exposure Assessment

The objective of the exposure assessment is to estimate the magnitude, frequency, duration, and routes of current and reasonably anticipated future human exposure to COPCs associated with the eight miles of the Lower Passaic River. The exposure assessment is based on the receptor scenarios that define the conditions of exposure to site-related COPCs. The exposure assessment evaluates cancer risks and non-cancer health hazards to a reasonably maximally exposed (RME) individual and a central tendency exposure (CTE) to describe the magnitude and range of exposure that might be incurred by the receptor groups. USEPA (1989) defines the RME as the highest exposure that is reasonably expected to occur at a site. According to USEPA guidance (1998), central-tendency estimates are intended to reflect central (more typical) estimates of exposure or dose. The objective of providing both the RME and CTE exposure cases is to set boundaries for the risk estimates, although decisions are based on the RME consistent with the NCP (USEPA, 1985).

5.1.1 Exposure Pathways

Consumption of fish and shellfish is anticipated to be the primary exposure pathway. For purposes of establishing current risks and comparing the relative risk reductions, cancer risks and non-cancer health hazards are estimated using exposure assumptions provided in the PAR (Battelle, 2006a) specifically for the adult/adolescent angler/sportsman and the young child (0 to 6 years) who may consume fish/crabs caught by a parent. These pathways will be included in the baseline HHRA for the site.

5.1.2 Exposure Media

5.1.2.1 Fish

To account for possible species preferences in human consumption of fish, a review of available published information was conducted to evaluate whether different species are preferentially targeted for consumption by anglers in the Lower Passaic River Study Area. Information reviewed included fishing licenses (NJDEP's E-Fishing Log Program that helps identify which fish are targeted [NJDEP, 2006a]), angler surveys, and other published information obtained for the study area. Table 5-1 summarizes fish species of the lower six miles of the Passaic River.

For purposes of this risk evaluation, fish species common to the lower portion of the river and species commonly eaten as reported in angler/creel surveys and published literature were identified and further

evaluated for use in determining the dataset to use for development of the EPCs. The EPCs for fish tissue-residue samples are based on a composite of tissue samples, rather than a single species, from those species that are of recreational importance (*i.e.*, may be appreciably consumed by recreational anglers/sportsmen). A review of the Passaic River Study Area Creel/Angler Survey (Desvousges *et al*., 2001) in conjunction with the fish community data collected by TSI, (2002) in accordance with the Passaic River Study Area Ecological Sampling Plan (TSI) (1999) resulted in the identification of target fish species for the Lower Passaic River. The identified fish species for this risk assessment include the white perch (predatory) and the American eel (bottom feeder of crabs, fish, and crayfish). In addition to being commonly caught and abundant in the study area, the white perch and American eel have been selected to represent two distinct ecological groups of fish: predators and bottom-feeders. This allows for the assessment of a variety of habitats, feeding strategies, and physiological factors that might result in differences in the uptake of contaminants. For instance, bottom-feeding species may bioaccumulate high contaminant concentrations from direct physical contact with contaminated sediment or by consuming epibenthic organisms and benthic invertebrates that live in contaminated sediment. Predator species are good indicators of persistent contaminants, such as mercury, which may be biomagnified through several trophic levels of the food web.

The Creel/Angler Survey (Desvousges, *et al*., 2001) identified the white perch and American eel as the most commonly caught fish at 65% and 17%, respectively. Striped bass, catfish (no specific species), and carp each comprised 7% of the catch (Desvousges *et al*., 2001). The fish community survey (TSI, 2002) identified striped bass, American eel, and white perch as being present throughout the lower seven-mile study area. The most common species identified in the lower eight miles of the Passaic River were inland silverside, mummichog, and Atlantic menhaden; none of which are species of interest for anglers. These three fish species are relatively small (up to 5 inches) and therefore would be of limited interest for human consumption, but they are forage fish for the bigger game fish. Other species that were not as prevalent as the forage fish, but identified in the fish community data survey are the striped bass and white perch. American eel were observed, but not in over abundant quantities. White catfish, channel catfish (more common than white catfish), and carp were similarly identified at low percentages. Based on a review of the Third River Watershed Document (Clifton Health Department/Clifton Environmental Protection Commission, 1999), white perch, striped bass, and American eel were also found in the upper reaches of the river (RM 8 to RM 17).

Average concentrations derived from the historical data for the four most common species identified (*i.e.*, white perch, American eel, striped bass, and brown bullhead [representing the only "catfish" data available in the historical data collected for the Passaic River]) have been plotted for some of the COPCs and are shown on Figures 5-1 and 5-2. White perch and striped bass represent predatory fish, whereas the American eel and the brown bullhead represent bottom feeders (American eel as a bottom feeder of crabs, fish and crayfish). Comparisons of average concentrations of TCDD TEQs are shown on Figure 5-1 for each of the fish species. Average concentrations are highest in the white perch and lowest in the American eel. Similarly, average concentrations of total PCBs are highest in the white perch (Figure 5-2), but lowest in the brown bullhead. Average concentrations of mercury and total DDx are fairly consistent among the four species (Figure 5-2).

Based on the consumption data from the Creel/Angler Survey (Desvousges *et al.*, 2001), the community surveys, and the extent of the historical analytical data available for each of the fish species, the white perch and American eel data (representing the upper and lower bounds of fish concentrations), were selected to derive an equal-weighted average concentration to represent the EPC for fish, similar to the methodology used in the Hudson River Risk Assessment (TAMS Consultants and Gradient Corp., 2000). These two fish species also represent the most commonly reported fish species consumed (Desvousges *et al*., 2001) in the study area. Historical data for the brown bullhead were not as abundant as that for the American eel, and because this particular species of catfish was not identified in any of the surveys as

being caught and consumed, data for this species were not included in the dataset to derive the EPC. Although the amount of historical data for the striped bass was similar to the white perch, concentrations of dioxins and PCBs were higher in the white perch, and white perch is caught and eaten more frequently than striped bass (Desvousges, *et al*., 2001). Therefore, data for the white perch, rather than the striped bass were used to provide a more conservative weighted average concentration for deriving the EPC. In the absence of site-specific data to support percent species intake, equal intake of the two representative species is assumed. The RI/FS will include further analysis of the fish species preferences and consumption patterns.

Fish tissue data from the white perch and American eel may overestimate or underestimate exposure to some of the COPCs as shown on Figures 5-1 and 5-2. For bioaccumulative contaminants, tissue concentration should be strongly related to age; however, because data used for this HHRE were not identifiable by age or weight classifications, it is uncertain if the data are representative of consumable fish. This uncertainty will be addressed in the RI/FS. In addition, excluding other species (*e.g.*, striped bass, catfish, and carp) known to be present in the river and reported as being caught less frequently and kept (Desvousges *et al*., 2001) adds to the uncertainty of the EPC, which will be addressed in the uncertainty section and will be further evaluated during the RI/FS.

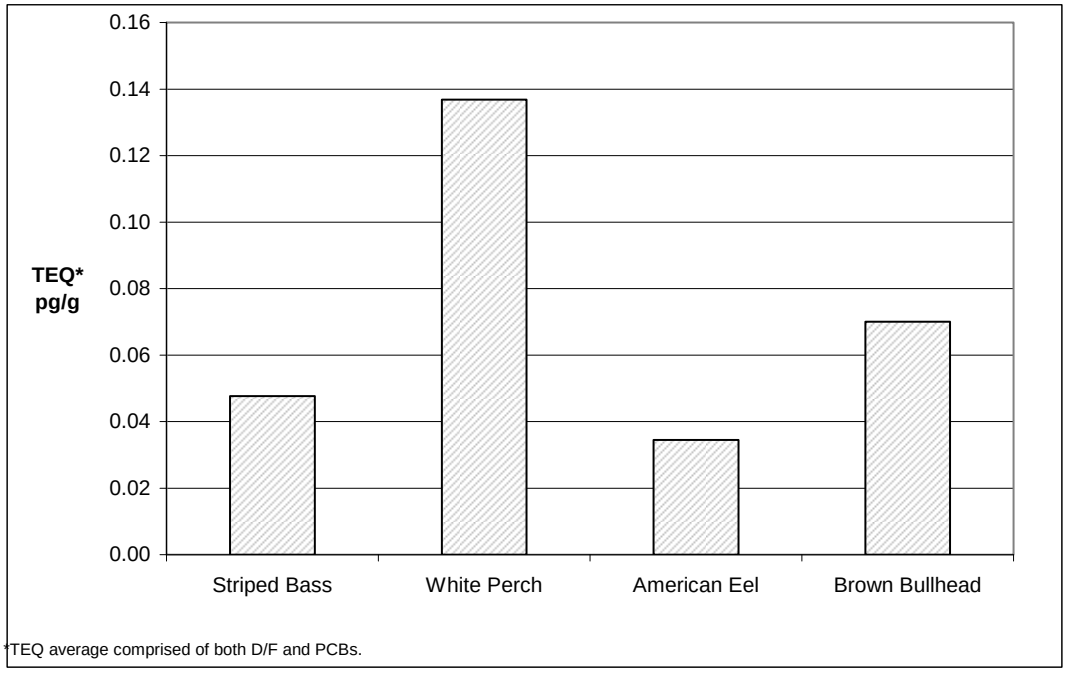


Figure 5-1. Comparison of Average Total TCDD TEQ Concentrations in Fish Tissue Samples.

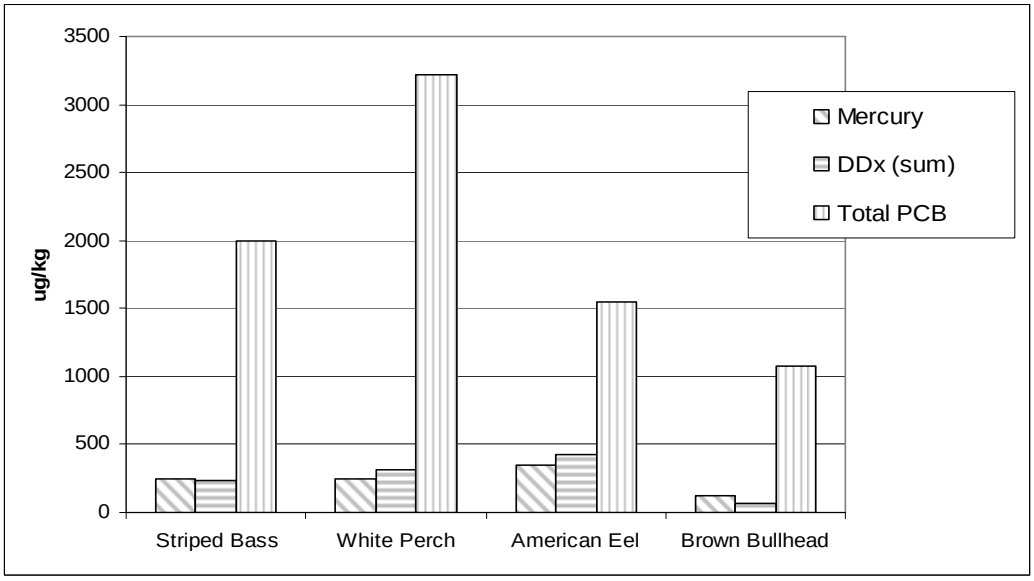
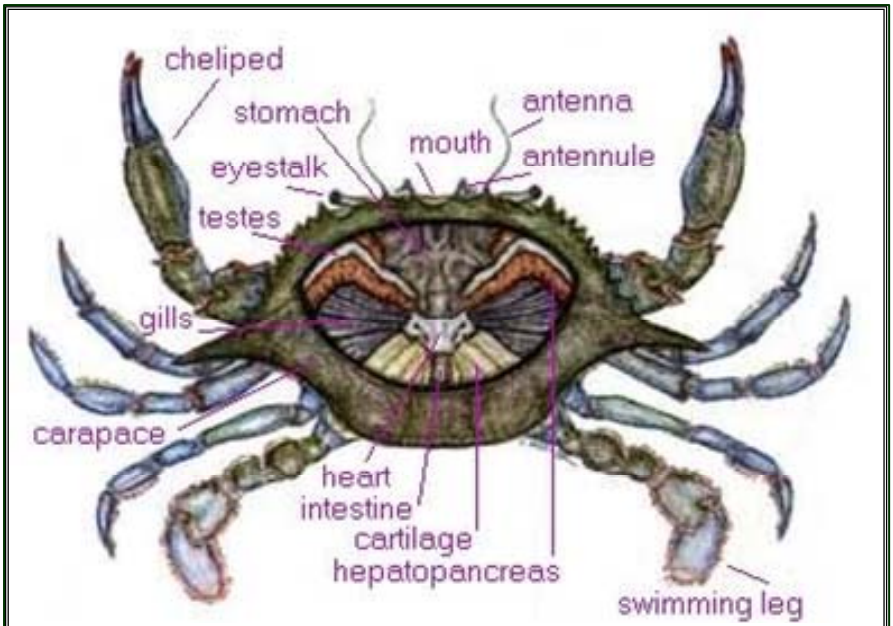


Figure 5-2. Comparison of Average Mercury, Total DDx, and Total PCB Concentrations in Fish Tissue Samples.

5.1.2.2 Crab

For crab, only the Blue crab is of interest in the study area. However, the part of the crab consumed is the primary concern for exposure because the amount of chemical with which an individual comes into contact depends on which parts of the crab are consumed. The highest levels of most chemical contaminants are found in the hepatopancreas (NJDEP, 2002), commonly known as the tomalley or green gland, which is the yellowish-green gland under the gills. The Blue crab anatomy is depicted on Figure 5-3. Information obtained from published literature report that individuals catching and consuming crab (*i.e.*, crabbers) may consume the edible white meat (or muscle), which includes the thoracic, claw, leg, and tail meat, and the hepatopancreas (Belton, *et al.*, 1985; May and Burger, 1996; NJDEP, 2002). Belton *et al.* (1985) stated that all of the crab tissues are considered edible food, whereas May and Burger (1996) and NJDEP (2002) report that only a small percentage of individuals purposefully consume the hepatopancreas. May and Burger (1996) reported that most crabbers in the Newark Bay Complex ate only cleaned crabs (hepatopancreas discarded), with fewer than 3% eating the whole crab. NJDEP (2002) reported that 15% of the population they surveyed in the Newark Bay Complex ate the hepatopancreas.



Source: Virginia Institute of Marine Science (http://www.marine-ed.org/bridge/bluecrabworkshop2.pdf)

Figure 5-3. Anatomy of a Blue Crab.

Comparisons of chemical concentrations found in muscle tissue and hepatopancreas samples have been reported in the literature. Belton *et al.* (1985) performed a differential analysis of the muscle and hepatopancreas samples for PCBs and organochlorine pesticides which indicated that both the PCBs and pesticide concentrations were much higher in the hepatopancreas samples (refer to Table 2C in Belton *et al.* 1985). Although Belton *et al.* (1985) did not specifically report the mean concentrations for the pesticide compounds, they did report the mean PCB concentrations in the hepatopancreas and muscle tissue as 6,520 µg/kg and 130 µg/kg, respectively. NJDEP (2002) summarized mean dioxin (as 2,3,7,8-TCDD TEQ) concentrations, originally reported in Skinner et al. (1997), as 0.19 µg/kg for the hepatopancreas samples (n=6) and 0.008 µg/kg for the muscle samples (n=6). In addition, NJDEP (2002) summarized the mean concentrations of 2,3,7,8-TCDD in hepatopancreas and muscle samples from a field sampling study conducted by Chemical Land Holdings, Inc. (CLH, 1999) as 0.262 µg/kg and 0.018 µg/kg, respectively. Therefore, based only on the analytical results for the two sample types, it can be assumed that an individual who only consumes the muscle tissue will be exposed to a smaller amount of the chemical versus someone who eats the hepatopancreas as well as the muscle tissue, unless cooking practices are considered as discussed below.

Exposure to the contaminant not only depends on the specific part of the crab the consumer eats, but on the method of cooking. NJDEP (2002) acknowledges that even those consumers who do not deliberately eat the hepatopancreas, are likely to be exposed to all or part of its content due to its fluid nature and its dispersion in the cooking liquid. Both Belton *et al.* (1985) and May and Burger (1996) state that boiling was the preferred method of cooking crabs of the individuals surveyed. Because the crab is cooked whole, consumption of only the muscle tissue would still result in exposure to the contaminants initially contained in the hepatopancreas. Although the State of New Jersey prohibits catching and consuming crabs from the Lower Passaic/Newark Bay Complex, the NJDEP guidance for consumption of fish and

crab (NJDEP, 2006b) provides crab preparation methods for those crabs obtained outside this region. NJDEP (2006b) states that there is no specific cooking method available to reduce the chemical contaminant levels in Blue crabs, and offers the following steps for proper preparation:

- Do not eat the green gland (hepatopancreas).
- Remove green gland (hepatopancreas) before cooking.
- After cooking, discard the cooking water.
- Do not use cooking water or green gland (hepatopancreas) in any juices, sauces, bisques or soups.

As evidenced in the published literature and addressed in the NJDEP guidance for consumption of fish and crab (2006c), even if the consumer does not eat the hepatopancreas, exposure to the chemical contaminant may still potentially occur if the crab is cooked before the hepatopancreas is removed and if the liquid used to boil the crab is used in juices, sauces, bisques, or soups.

For the purposes of this risk evaluation, exposure to COPCs in the hepatopancreas and muscle is anticipated based on crab cooking practices. Therefore, analytical results for both types of tissue samples will be combined and used to determine the EPC for crab consumption, similar to the composite sample approach described in NJDEP (2002). The uncertainties associated with an EPC derived using a composite hepatopancreas/muscle approach are addressed in the uncertainty section because this approach may under- or over-estimate total risk.

Table 5-1. Fish Species Located Within the Lower Six Miles of the Passaic River.

Sampling Locations Within the Lower 6 Miles [1,2]
Common carp [1]
Channel catfish [1]
Bluefish [3]
Blue crab [3,4]
American eel [1]
Striped bass [1]
White perch [1]
Atlantic menhaden [1]
Brown bullhead [1]
Weakfish [2]
Gizzard shad [2]

(1) ChemRisk, 1995.
(2) Iannuzzi and Ludwig, 2004.
(3) NJDEP, 2006b.
(4) Desvousges *et al.,* 2001.

5.1.3 Potential Receptors and Exposure Routes

The angler/sportsman is defined as an adult individual catching and consuming a variety of fish (*e.g.*, carp, striped bass, catfish, and American eel), and other local species (*e.g.*, Blue crab) from the river. The adolescent, aged 10 to 18 years, evaluated in this survey is another possible angling population that may fish/crab and consume their catch. This information is based on studies of angling activities that have found children typically begin fishing at about the age of 10 years (USEPA, 2000a). In addition, many states with licensing programs require children to have licenses beginning at the age of 16 years before

they can legally fish (NJDEP, 2006c). Young children (0 to 6 years) are assumed to consume fish caught by their angling parent. The collection and consumption of fish and shellfish from the Passaic River has been well documented in a creel survey conducted by Belton *et al*. (1985) for NJDEP, as well as in other published literature regarding angler's perception of risk from contaminated fish (May and Burger, 1996; Burger *et al*., 1999; Kirk-Pflugh *et al*., 1999); therefore, it is clear that this exposure pathway is complete for the Angler/Sportsman.

In addition to the above-mentioned routes of exposure, other potential pathways exist by which individuals may be exposed to COPCs in the Lower Passaic River. One such a pathway is exposure from eating game (*e.g.*, turtles, waterfowl) also found along the banks of the Lower Passaic River. Snapping turtles and water fowl may contain high concentrations of dioxins and PCBs in their fat and internal organs. Although public health advisories for consumption of these animals have not been issued by NJDEP, two neighboring states, Pennsylvania and New York, have issued consumption advisories for certain game (New York State Department of Health [NYSDH], 2006; Pennsylvania DEP, 2006) associated with the presence of PCBs in their state waterways. However, because there are no historical data on chemical concentrations in the tissues of these organisms, consumption of waterfowl, turtles, and other species is not addressed in this HHRE quantitatively but rather qualitatively as an area of uncertainty. For individuals who consume these animals in addition to fish and crab, risks would be expected to be higher.

It is assumed that an adult and/or adolescent angler/sportsman shares his/her catch with an adolescent (age 10-18 years) and a child (age 0-6 years) family member. Although typically young children and adolescents under age 15 are not required to have fishing licenses, several sources indicate that many children consume sport-caught freshwater fish (Connelly *et al.*, 1990; Connelly *et al.*, 1992; Wendt, 1986). Subpopulations of highly exposed or less-exposed anglers have not been explicitly characterized, but instead are assumed to be represented in the overall fish ingestion rate and this will be further evaluated in the RI/FS baseline HHRA. The potential exists, however, that, distinct subpopulations may fish in the 7-mile study area and consume higher amounts of fish, but are not explicitly identified in the creel surveys used in this analysis. There is some degree of uncertainty as to whether these subpopulations have been adequately addressed in this risk evaluation. Subsistence fishing was not evaluated HHRE, but may be evaluated in the RI/FS after further analysis of the creel surveys.

Other potential exposure pathways relevant to the adult angler/sportsman, as indicated in the CSM (Figure 4-1), include direct exposures (*i.e*., dermal contact and incidental ingestion) to sediments and surface water contacted during collection activities. Because consumption of biota (fish and crab) is anticipated to be a risk driver it is the only pathway evaluated in this assessment. The other pathways and potential higher end ingestion rates will be further evaluated in the RI/FS. Omitting other applicable exposure pathways and higher end ingestion rates leads to an underestimate of risk, as discussed in Section 5.4.

5.1.4 Estimation of Chemical Intake

Intake is estimated by combining EPCs with the variables that describe exposure:

- Rate of contact with the medium;
- Frequency of contact;
- Duration of contact; and
- Body weight of the exposed individual.

Chemical intake from ingesting fish is estimated following USEPA (1989) guidance and other applicable guidance, guidelines, and policies. An intake factor is the amount of a chemical in a quantity of a

medium (*e.g.*, fish tissue) taken into the body through an exposure route (*e.g.*, ingestion) and available for absorption. It is expressed in units of milligram (mg) of chemical per kilogram (kg) body weight per day (mg/kg-day). Intake of a chemical that results in carcinogenic effects is calculated by averaging the dose over a lifetime (70 years x 365 days/year) (USEPA, 2005b). The intake factor for carcinogenic effects is termed the lifetime average daily dose (LADD). Intake of COPCs that produce non-cancer health effects is averaged over the period of exposure [exposure duration (ED) x 365 days/year]. The intake factor for exposure durations equal to or longer than seven years is termed the chronic average daily dose (ADD) (USEPA, 1989). Intake will be estimated for LADD and ADD ingestion of fish and crab for an adult, adolescent, and child (0 to 6 years) with appropriate adjustments for ingestion rates and bodyweights.

The equation used to calculate the LADD/ADD for ingestion of biota (fish and crab) is provided as:

$$LADD / ADD = \frac{C_t xIRxEFxFIx(1 - Loss)xED}{BWxAT} \qquad (5\text{-}1)$$

where:

C_t = Biota tissue concentration (mg/kg)
ED = Exposure duration (years)
EF = Exposure frequency (days/year)
IR = Annualized ingestion rate (kg/day)
FI = Fraction from contaminated source (unitless)
Loss = Cooking loss (g/g)
BW = Body weight (kg)
AT = Averaging time (days) - period over which exposure is averaged (days); over a lifetime for evaluating cancer risks and over the appropriate exposure duration for evaluating non-cancer health hazards.

5.1.4.1 Exposure Point Concentrations

As explained in Section 4.2.3, EPCs for COPCs in fish and crab were calculated as the 95% UCL of the arithmetic average following guidance in USEPA (2002a). For completion of this risk evaluation, the EPCs used in the RME and CTE evaluations are the same. Note that the EPCs are assumed to remain constant in fish/crab throughout a lifetime and do not consider any attenuation or degradation of the chemical in sediment that may occur over time. The RME and CTE exposures differ with regard to the receptor-specific exposure variables, which are further described below and summarized in Appendix C (Tables C-5 through C-10). The EPCs for each of the COPCs in fish and crab are presented in Table 4-1. Consistent with USEPA RAGS Part D guidance (2001), the EPCs are also presented in the risk assessment tables provided in Attachment C, Tables C-1 and C-3 for the fish RME and CTE, respectively, and in Attachment C, Tables C-2 and C-4 for the crab RME and CTE, respectively.

As described in Section 5.1.2.1, white perch and American eel were identified as the fish species that potential receptors are more likely to catch and eat from the Lower Passaic River. As such, the historical analytical data for white perch and American eel collected throughout the lower eight miles of the Lower Passaic River were combined for each of the COPCs and used to determine the EPCs to evaluate exposures associated with consumption of fish. Fish tissue data were noted as "whole organism" which is assumed to include skin, organs, and the head. Specific data for fillet samples were not available for this HHRE, and therefore comparisons of concentration differences between fillet and whole organism samples could not be prepared. Therefore, EPCs derived for COPCs in fish may be overestimated because whole organism samples were used rather than fillet samples.

Historical crab analytical data were used separately to determine the EPCs for ingestion of crab. The historical crab data set consists of three tissue sample types: muscle, hepatopancreas, and all soft tissue (*i.e.*, all tissue excluding the hepatopancreas). The muscle sample type comprises roughly 50% of the dataset, whereas the hepatopancreas and the soft tissue sample types make up 20% and 30%, respectively. All historical crab tissue data, including analytical results from different investigations and different sample types, were combined for each of the COPCs and used to determine the EPCs representative of the entire eight miles of the Lower Passaic River. As described in Section 5.1.2.2, higher concentrations of contaminants are usually found in the hepatopancreas rather than the muscle tissue. In order to demonstrate the concentration differences among the sample types, comparisons of the average concentrations observed for the three types of samples collected for crabs have been provided on Figures 5-4 through 5-7 for dioxins, total PCBs, total DDx, and mercury. The data used for these comparisons are a subset of the historical crab dataset that were collected by Chemical Land Holdings, Inc. in 1999/2000 in accordance with an USEPA-approved Ecological Sampling Plan (CLH, 1999). The CLH data have been chosen to demonstrate concentration differences among sample types because these data are comprised of 15 co-located samples of the hepatopancreas, muscle, and all soft tissue (*i.e.*, edible tissue minus the hepatopancreas).

Figures 5-4 through 5-7 show comparisons of the average concentrations observed for the three types of samples collected from crabs to demonstrate the range of concentrations for each sample type. As shown on these figures, concentrations associated with the hepatopancreas samples are much higher for the organic compounds than those for the other sample types, similar to what Belton *et al*. (1985) observed with their data. Conversely, mercury concentrations are higher in the muscle tissue as shown on Figure 5-7.

The EPC used in this risk evaluation was derived by combining all of the sample results, analogous to the "All Soft Tissue + Hepatopancreas" values shown on Figures 5-4 through 5-7. An EPC that has been derived by compositing the sample types therefore may be more representative for those consumers who do not deliberately eat the hepatopancreas, but are likely to be exposed to all or part of its content as a result of how the crab is cooked as described in Section 5.1.2.2. Conversely, the EPC may be over- or underestimated for those individuals specifically only eating the hepatopancreas or muscle tissue.

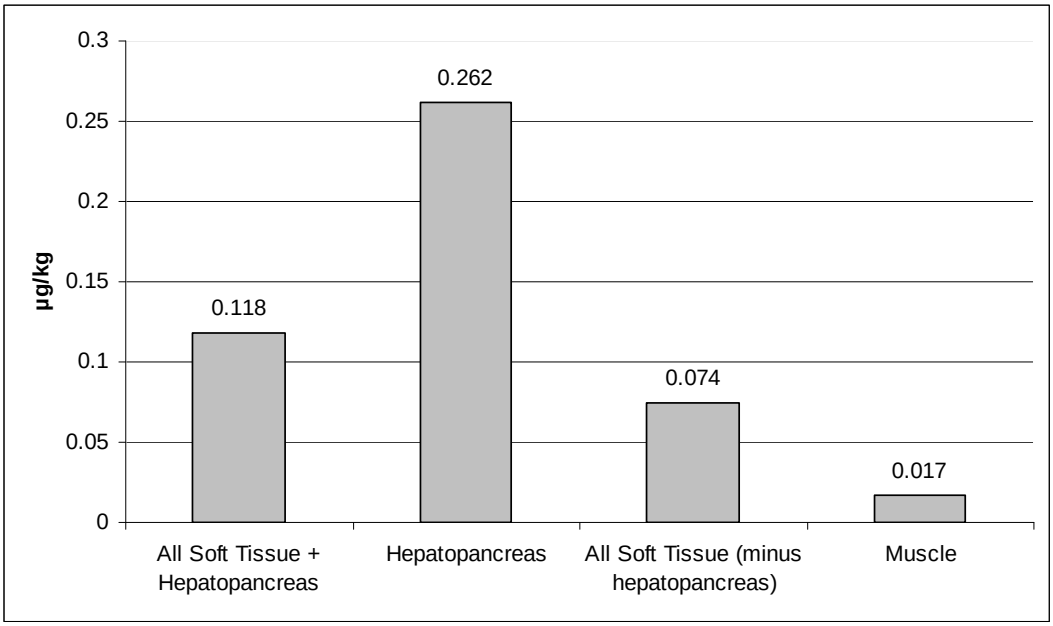


Figure 5-4. Comparison of Average 2,3,7,8-TCDD Concentrations Among Crab Sample Types.

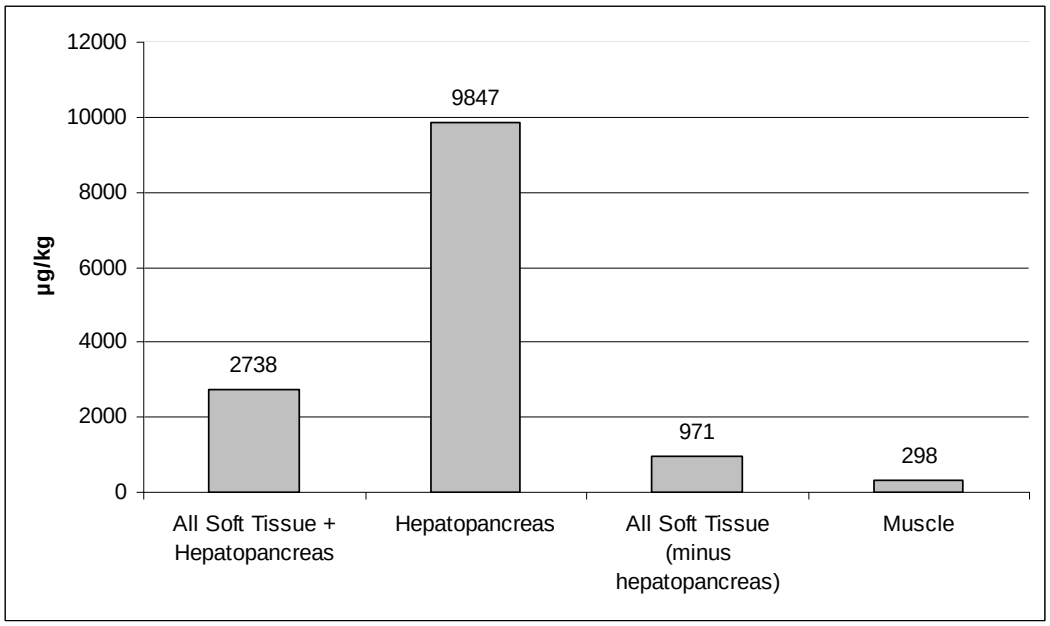


Figure 5-5. Comparison of Average Total PCB Concentrations Among Crab Sample Types.

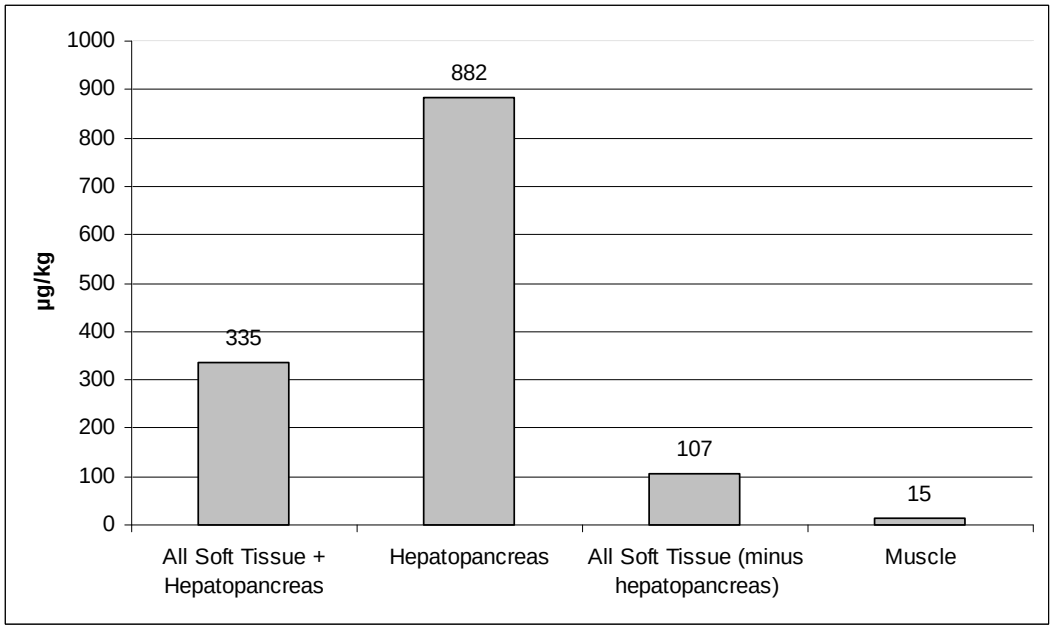


Figure 5-6. Comparison of Average Total DDx Concentrations Among Crab Sample Types.

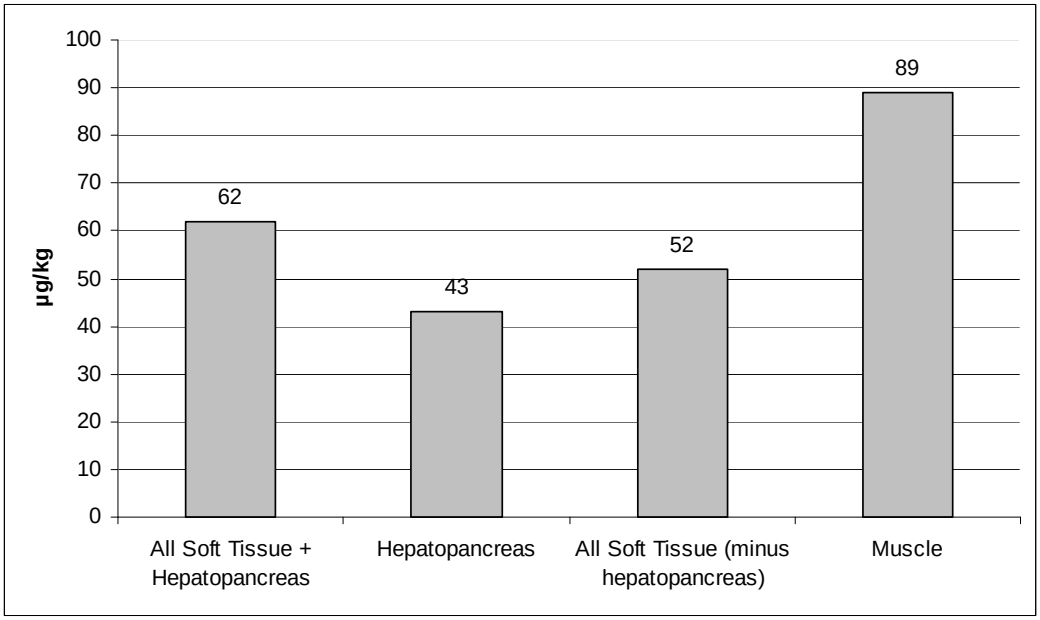


Figure 5-7. Comparison of Average Mercury Concentrations Among Crab Sample Types.

For comparison purposes, NJDEP routine monitoring data has been summarized and presented in Table 5-2. In 2004, Blue crabs were collected from the Lower Passaic River and Newark Bay Complex by Dr. Richard Horwitz under the statewide "Routine Monitoring Program for Toxics in Fish" as developed by NJDEP's Division of Science, Research and Technology (DSRT) (NJDEP, 2006d). The objective of the program is to update human health consumption advisories for fish and shellfish of concern as well as identify contaminant concentrations in marine and estuarine species along New Jersey's coast. Muscle meat and hepatopancreas composite tissue samples from five Blue crabs collected specifically from the Lower Passaic River were analyzed for total PCBs, total DDx, total mercury, and 2,3,7,8-TCDD. Average concentrations of total PCBs and total DDx compounds determined by NJDEP for hepatopancreas and muscle tissue samples were much lower than those associated with the site data (see Figures 5-5 and 5-6, respectively). Conversely, NJDEP data for the average total mercury concentrations in hepatopancreas and muscle samples were more than double those concentrations determined for the site (Figure 5-7). Hepatopancreas and muscle tissue samples for both NJDEP and the site were consistent (Figure 5-4).

5.1.4.2 Exposure Factors

The population of concern in this HHRE consists of the inhabitants of the towns, cities, and rural areas surrounding the 8-mile stretch of the Passaic River who may fish and/or crab in the river or eat catch from this area. The angler population is defined as those individuals who consume self-caught fish from the 8-mile stretch of the Passaic River regardless of the fish/crab consumption advisories. The assessment of fish consumption by the angler population includes young children (ages 0-6), adolescents (ages 10 to 18 years), and adults (over 18). Prenatal and neonatal exposures are evaluated qualitatively.

Table 5-2. Summary Statistics for Blue Crab Contaminants[(a)].

Analyte	Tissue	Count	Concentration (µg/kg wet weight)		
			Minimum	Maximum	Average
Total PCBs	Hepatopancreas	5	1,668	7,020	3,597
	Muscle	5	48.7	97.3	70.3
	Combined	10	48.7	7,020	1,834
Total DDx	Hepatopancreas	5	263	1182	596
	Muscle	5	14.5	22.9	18.1
	Combined	10	14.5	1,182	307
Total Mercury	Hepatopancreas	5	60.0	100	86.0
	Muscle	5	150	210	182
	Combined	10	60.0	210	134
2,3,7,8-TCDD	Hepatopancreas	5	0.175	0.394	0.288
	Muscle	4[(b)]	0.009	0.013	0.011
	Combined	9[(b)]	0.009	0.394	0.165

Note: Data are draft and subject to change.
(a) Source: NJDEP, 2006d.
(b) One result was not detected and the method detection limit (MDL) was not available.

The specific exposure parameter values proposed for estimating intake for the RME and CTE for ingestion of fish are presented in RAGS Part D tables (USEPA, 2001) in Attachment C, Tables C-5 through C-7 for the adult, adolescent, and child receptors, respectively. Similarly in Attachment C, Tables C-8 through C-10 present the specific exposure parameter values proposed for estimating intake for the RME and CTE for ingestion of crab for the adult, adolescent, and child receptors, respectively. A description of each of the key exposure parameters and the rationale for their selection is provided below.

Self-Caught Ingestion Rates of Fish (IR_f)

The ingestion rate is the amount of fish an individual consumes on a daily basis in units of grams per day based on averaging the reported consumption rate in one year per 365 days per year. The ingestion rate assumes the fish are caught while angling from the Lower Passaic River only. It is expected that ingestion of fish from other sources would add to the amount an individual ingested annually. Ingestion rates for fish have been annualized and are presented in grams eaten per day (g/day).

For consumption of fish, ingestion rates based on data collected for recreational freshwater anglers were obtained from the Exposure Factors Handbook (EFH) (USEPA, 1997). For the adult angler/sportsman, 25 g/day, which is the 95th percentile, was used for the RME, whereas the recommended mean of 8 g/day was used for the CTE. The values in the EFH are based on fish ingestion studies from several different freshwater locations within the country. The surveys include: 1992 Maine Angler Survey (Ebert *et al.*, 1993), 1992 Lake Ontario Diary Study (Connelly *et al.*, 1996), and 1989 Michigan Sport Angler survey (West *et al.*, 1989). The ingestion rate for fish and crab identified in the Burger survey (2002) found that 8 to 25% of the population ingested 1,500 g/day which is equivalent to 50% from fish and 50% from crabs (as discussed below for the crab ingestion rate).

Ingestion rates for the adolescent and child were based on the assumptions that the intake for the adolescent will be approximately 2/3 that of the adult, whereas the intake for the child will be approximately 1/3 that of the adult portion (USEPA, 1997). This assumption is based on the fish consumption rates provided in Table 10-1 of the EFH (USEPA, 1997) for a child aged 0 to 9 years, an

adolescent from 10 to 19 years of age, and an adult aged 20 to 70+ years of age (intake averaged over the six adult age groups). According to Table 10-1 of the EFH (USEPA, 1997), the 95th percentile intake for children aged 0 to 9 years is 16.5 g/day. For adolescents aged 10 to 19 the 95th percentile intake in USEPA's EFH is 26.8 g/day. The selected ingestion rates are consistent with those presented in the EFH considering the specific ages of the populations being evaluated in this survey and also within the upper bounds of the ingestion rates at the 90th percentile or above (USEPA, 1997). Thus, for the RME, ingestion rates of 8 g/day and 17 g/day are used for the child and adolescent receptors, respectively. For the CTE, the ingestion rates of 3 g/day and 5 g/day are used for the child and adolescent receptors, respectively.

Self-Caught Ingestion Rates of Crab (IR_c)

The ingestion rate is the amount of crab an individual consumes on a daily basis in units of grams per day based on averaging the reported consumption rate in one year per 365 days per year. The ingestion rate assumes the crabs are caught while angling from the Lower Passaic River only. It is expected that ingestion of crab from other sources would add to the amount an individual ingested annually. Ingestion rates for crab have been annualized and are presented in grams eaten per day.

There is limited information in the published literature regarding the consumption rates of crabs. Studies conducted in the Newark Bay Complex area were reviewed (Burger, 2002; Burger *et al.,* 1999; and May and Burger, 1996) to identify an appropriate consumption rate. Of the studies reviewed, the Burger study (2002) was the only one that contained sufficient information regarding crab consumption in the area of the Lower Passaic River, which was used to derive a consumption rate for this risk evaluation.

In 1999, a published study by Burger *et al.* included interviews with 267 people angling at several locations within the Newark Bay Complex, including parts of the Passaic River, on a regular basis between May 15 and September 15. The survey included questions regarding the consumption pattern of the individual who was fishing and/or crabbing, along with questions for demographics, knowledge of advisories, and reasons for angling. Results of the study indicated that there were no ethnic differences (Asian, Hispanic, Black, White) in the percentage of people who crabbed, nor were there ethnic differences in age, annual income, or health ratings. Burger *et al*. (1999), did however, identify differences in consumption patterns across the various ethnic groups. They found that consumption increased with the angler's age, and decreased with income, and noted that Asians ate few crabs and mainly fish, while the other ethnic groups ate mainly crabs. Overall, 49% of Whites did not eat their catch, while 40% of Hispanics, 24% of Asians, and 22% of Blacks did not eat their catch. In addition, a higher percentage of Blacks and Hispanics reported eating more of their catch (fish, crab, or both) per month than Whites and Asians.

A yearly consumption rate for self-caught crab was developed by Burger (2002), by multiplying the number of crab meals eaten per month by the number of crabs eaten at each meal by the number of months per year crabs are caught, assuming the average serving size from one crab is 70 grams. Crab consumption patterns for people surveyed were determined for two groups of individuals: 1) for people that only catch crab; and 2) for people that catch both crab and fish. Burger (2002) notes that the majority of people interviewed mainly fished or mainly crabbed, and that more than 30% of the people who fished and crabbed in the Newark Bay Complex did not eat their catch. However, the study also reports that 8% to 25% of the people ate more than 1500 g/month of self-caught fish and crab. Table 5-2 summarizes the crab consumption patterns for people that crab only and for those that both crab and fish. Note that people reported crabbing only three months out of the year and only data from this three month period was used to calculate the annual ingestion rate. This may potentially underestimate the risks and hazards.

For purposes of this risk evaluation, consumption of crab and fish were assumed to occur in separate populations so that people either ate fish or ate crab, but not both. This approach may potentially

underestimate risks for those individuals who both consume fish and ingest crabs. As shown in Table 5-3, individuals that both fish and crab reported eating more crab per year than those that only crabbed. The uncertainty associated with assuming individuals did not eat both fish and crab is further addressed in the uncertainty Section 5.4 "Uncertainty Analysis".

Table 5-3. Crab Consumption Patterns for Consumers Surveyed in the Newark Bay Complex in 1999.[a]

Parameter	Consumers of Crab Only	Consumers of Both Crab and Fish
Sample size (n)	110	33
Number of times per month they eat self-caught crabs	3.39 ± 0.42	2.96 ± 0.45
Number of self-caught crabs (*i.e.,* serving size)	6.15 ± 0.85	7.27 ± 0.91
Amount of self-caught crabs for each serving (g)	439 ± 61.2	509 ± 63.8
Monthly consumption of self-caught crabs (g)	1,980 ± 561	1,620 ± 330
Number of months per year they crab	3.31 ± 0.13	3.50 ± 0.37
Yearly consumption of self-caught crab (g)[b]	5,760 ± 1,360	6,230 ± 1,790

Source: Burger, 2002 (Table 2).

a. Values provided are means ± standard errors based on computed yearly consumption for each person individually; therefore, yearly consumption values provided in the table are not exactly reproducible.

b. Assumes average weight of meat from crabs is 70 g.

Based on the crab consumption patterns for people that crab only, as reported in Burger (2002), the RME ingestion rate for the adult angler/sportsman was selected as 23 g/day. This value is the 95% UCL of the yearly consumption value, derived as follows:

$$95\%UCL = \frac{5{,}760\,\frac{g}{year} + \left(1.96 \times 1{,}360\,\frac{g}{year}\right)}{365\,\frac{days}{year}} \qquad (5\text{-}2)$$

Although Burger (2002) did not identify the distribution of the data, the data were assumed to be normally distributed based on the central limit theorem. This states that sampling distribution means tend toward normality as n gets large. In this particular case, n=110, which justifies the use of procedures based on the normal distribution even if the underlying population is not normal (McBean and Rovers, 1998).

The average yearly consumption rate of 5,760 g/year (16 g/day) was selected as the adult CTE ingestion rate. Ingestion rates for the child and adolescent receptors were estimated assuming rates of 1/3 and 2/3 that of the adult ingestion rate, respectively, as was assumed for fish ingestion.

Crab consumption data were obtained for the Newark Bay Complex area by NJDEP from an angler survey administered by NJDEP in 1995 (NJDEP, 2002). Based on the results of this survey, 65% of the population surveyed consumed self-caught crab once per week or less whereas 28% of the individuals reportedly consumed crab at least 2 to 3 times per week. The survey results indicated that the majority of surveyed individuals (56%) consumed between one and six crabs at each meal and seven crabs or more were eaten by only 35% of the population. NJDEP used this consumption information to estimate a range

of the amount of crab consumed per meal per day, assuming the edible mass of the crab was 75 g. Depending on the number of crab meals per day (*i.e.*, 1 crab meal/day or 0.14 crab meal/day) and the number of crabs eaten at each meal (*i.e.*, 2, 5, or 15 crabs), the amount of crab consumed per day ranged from 21 g/day up to 1,125 g/day. The consumption rate of 23 g/day derived from the Burger (2002) data is consistent with the lower value derived from the NJDEP survey data. The majority of the NJDEP surveyed population is most likely represented by this lower daily ingestion rate. However, for the small percentage of the population who consume a larger portion of crab, the risks/hazards are likely to be underestimated with the use of the lower ingestion rate, which is addressed as an uncertainty in the uncertainty analysis (Section 5.4).

Fraction from Contaminated Source (FI)

This factor is applied to account for possible exposures to contaminants from other sources with similar contaminants. This is particularly relevant for the site given the Lower Passaic River watershed consists of over 100 square miles of highly developed urban area that supports a large population of people. Although it is possible that an angler catches and consumes fish from other rivers in the area, this risk evaluation assumes that 100% of the catch is obtained from the Lower Passaic River. Therefore, an FI of 1 is used for the RME and CTE scenarios.

Cooking Loss (CL) for Fish

Preparation and cooking procedures can modify the amount of contaminant ingested by consumers, consequently, modifying exposure and dose. Several studies have been conducted in an attempt to quantify this modification and a variety of factors have been investigated including the species of fish, preparation method (*e.g.,* skin-on vs. skin-off), cooking method (baking, broiling, deep frying, *etc.*), fattiness of the fish sampled (within the same species), and water body where the fish were collected. The USEPA (2000a) summarized the percent reductions of organic contaminants resulting from preparation method, cooking method, species and location. The range of reduction percentages for the chemicals of potential concern are summarized in Table 5-4, with the exception of PCBs. These studies show wide ranges in the percentage of reduction for each of the chemicals investigated. Thus, it is challenging to

Table 5-4. Summary of Contaminant Loss from Fish Due to Cooking (Skin Off and Skin On).

Contaminant	Preparation Method	Percent Loss Value (%)	Study Reference
p,p'-DDD	Skin off	4 to 88	Zabik *et al.*, 1995a; 1996
p,p'-DDD	Skin on	10 to 54	Zabik *et al.*, 1995a
p,p'-DDE	Skin off	7 to 61	Zabik *et al.*, 1995a; 1996
p,p'-DDE	Skin on	16 to 59	Zabik *et al.*, 1995b
DDE	Trimmed	52 to 54	Skea *et al.,* 1979
p,p'-DDT	Skin off	1 to 80	Zabik *et al.*, 1994; 1995a; 1996
p,p'-DDT	Skin on	23 to 60	Zabik *et al.*, 1994; 1995a
DDT	Trimming/Skin off	1 to 62	Reinert *et al.,* 1972; Zabik *et al.* 1994
DDT	Skin on	4 to 16	Zabik *et al.*, 1994
Dieldrin	Skin off	4 to 88	Zabik *et al.*, 1994; 1995a;b; 1996;
Dieldrin	Skin on	3 to 93	Zabik *et al.*, 1994; 1995a;b
α-Chlordane	Skin off	3 to 63	Zabik *et al.*, 1994; 1995a; 1996
α-Chlordane	Skin on	(-)25 to 63	Zabik *et al.*, 1994;1995a
γ-Chlordane	Skin off	1 to 83	Zabik *et al.*, 1995a: 1996
γ-Chlordane	Skin on	20 to 50	Zabik *et al.*, 1995a
Chlordane Complex	Skin on	3 to 60	Zabik *et al.*, 1995b
TCDD	Skin off	~54 to ~57	Zabik and Zabik, 1995
TCDD	Skin on	~ 37 to ~80	Zabik and Zabik, 1995

Source: USEPA, 2000a.

select a reduction factor that can be applied for a particular chemical. Therefore, cooking loss values will be selected based on percent losses derived by combining all cooking methods, as well as USEPA default recommendations, which are further described below.

Summary statistics of the range of reduction percentages for the COPCs, as reported by the USEPA (2000a), are summarized in Table 5-5. Note that Table 5-5 summarizes the percent loss values for skin-on, skin-off, and combined (skin-off plus skin-on). Because there were no consistent differences in contaminant losses between cooking methods, the results were only grouped according to contaminant, and not by cooking method.

For this particular review of cooking loss, PCBs were not included because numerous studies regarding PCB cooking loss were evaluated in the HHRE for the Hudson River (TAMS/Gradient Corp., 2000). The 12 studies reviewed in the Hudson River HHRE regarding cooking loss found the rate of cooking loss ranged from 0 to 74% with most PCB losses between 10% and 40%. Based on the results provided in the Hudson River risk assessment, (USEPA, 2000a) a factor of 20% as the cooking loss factor for the CTE was used, noting that the value of 20% is midpoint between 0% and 40%. For the RME, 0% cooking loss is assumed.

Generally, chemical contaminants are not distributed uniformly in fish. Fatty tissues, for example, will concentrate many organic chemicals more readily than muscle tissue. For those chemicals that accumulate in the fatty tissues, removing the skin and fat that collects beneath the skin and along the lateral line will reduce contaminant exposure. Also, to make adjustments to dose accurately, it is important to match the dose modification factors to the type of sample from which the fish contaminant concentrations was measured. For example, it would not be appropriate to apply a modification factor based on removal of skin if the sample analyzed for contaminants was already a "skin-off" fillet.

The EFH (USEPA, 1997) provides a recommended default adjustment for cooking and preparation loss. The values given in the EFH for fish are 30% for mean net cooking loss (includes dripping and volatile losses during cooking, averaged over various cuts and preparation methods) and 11% for mean net post cooking loss (includes losses from cutting, shrinkage, excess fat, bones, scraps and juices, averaged over various cuts and preparation methods). The EFH recommends that the modified intake rates be calculated as:

$$I_A = I \times (1 - L_1) \times (1 - L_2) \qquad (5\text{-}3)$$

Where:

I_A = Adjusted Intake Rate
I = Intake Rate
L_1 = Cooking Loss
L_2 = Post-Cooking Loss

By applying the mean percent weight losses presented in the EFH, the adjusted intake rate is calculated as follows:

$$I_A = I \times (1 - 0.30) \times (1 - 0.11) \qquad (5\text{-}4)$$

$$I_A = I \times 0.7 \times 0.89 \qquad (5\text{-}5)$$

$$I_A = I \times 0.62 \qquad (5\text{-}6)$$

Table 5-5. Summary Statistics for Contaminant Percent Loss[a] from Fish Due to Cooking.

	Skin-Off					Skin-On						Combined[(b)]				
Contaminant [(c)]	Minimum	Average	50th Percentile	90th Percentile	Maximum	Minimum	Average	50th Percentile	90th Percentile	95th Percentile	Maximum	Minimum	Average	50th Percentile	90th Percentile	Maximum
DDD	4	30	19	61	88	10	37	36	54	54	54	4	31	30	58	88
DDE	7	30	27	52	75	7	39	39	49	54	59	7	32	35	52	75
DDT	0	38	30	69	141	4	33	29	58	59	60	0	37	30	64	141
Chlordane	1	29	30	51	83	3	38	38	52	57	63	1	32	33	51	83
Dieldrin	4	29	25	52	88	3	36	38	58	61	93	3	32	30	55	93
TCDD	54	56	56	57	57	37	51	44	69	75	80	37	53	49	69	80

Source: USEPA, 2000a

[a.] Percent losses are derived by combining all cooking methods.

[b.] Combined includes both skin-on and skin-off results.

[c.] Contaminants have all been grouped under one heading. For example, alpha chlordane and gamma chlordane have been combined and results summarized as "chlordane".

Thus, the total cooking loss and preparation adjustment amounts to 38% contaminant concentration reduction, which is similar to the values listed in Table 5-5 under the combined 50th percentile column heading. Note that the mean cooking loss percentages are based on averages over a variety of fish, including bass, bluefish, butterfish, cod, flounder, haddock, halibut, lake trout, mackerel, perch, porgy, red snapper, rockfish, salmon, sea trout, shad, smelt, sole, spot, squid, swordfish steak, trout, and whitefish.

In general, for heavy metals, tissue residues are not significantly reduced by processing or cooking methods. Therefore, preparation and cooking loss adjustments should not be applied for metals in most cases (USEPA, 2000a). Mercury, however, may be an exception. Mercury binds strongly to proteins and thus concentrates in the muscle tissue of the fish. It also concentrates in the liver and kidneys, although to a lesser extent (USEPA, 2000a). Several studies on the effects of preparation and cooking on mercury have shown that mercury concentrations are less in raw fish than in cooked fish, although the total amounts of mercury remain the same. The higher concentrations in cooked fish are attributed to the loss of liquid and fat during cooking which results in a higher concentration. Morgan *et al.* (1997) found that mercury concentrations in pan-fried, baked, and broiled walleye fillets and deep-fried and baked whitefish livers ranged from 1.1 to 1.5 times higher than corresponding raw portions. In lake trout, mercury concentrations were 1.5 to 2.0 times higher in smoked fish than in the raw portions. Burger *et al.* (2003) calculated preparation factors of 1.5 to 1.8 for deep fried large mouth bass. They concluded that based on these two studies, a preparation factor of 2 would be a suitable, protective default for estimating safe consumption levels.

The losses reported generally do not include an accounting for degradation of the contaminants. Until there is more information about the toxicity of the byproducts generated during the degradation of PCBs, dioxins/furans, organochlorine pesticides, or other chemicals of concern, USEPA recommends that no dose modification be assumed due to degradation alone (USEPA, 2000a).

Table 5-5 summarizes the range of cooking losses from fish that are examined in this risk evaluation. For RME, a cooking loss of 0% is proposed for all contaminants to be consistent with the PCB cooking loss. For CTE, the 50th percentile cooking loss percent value for combined skin-on/skin-off is used as shown on Table 5-6. For mercury, both the RME and CTE estimates are 0% which is in agreement with USEPA (2000a) which states preparation and cooking loss adjustments should not be applied for metals in most cases. The effect of cooking and mercury concentrations will be addressed further in the uncertainty section (Section 5.4).

Cooking Loss (CL) for Crab

Blue crabs are most often cooked whole by boiling or steaming (Sea Grant Marine Advisory Program, 2006). Exposure to the contaminant not only depends on the specific part of the crab the consumer eats, but on the method of cooking as previously discussed in Section 5.1.2.2. Zabik *et al*., (1992) looked at the changes in the distribution of PCBs in Blue crab caused by boiling or steaming and found that both cooking procedures reduced PCBs by more than 20% with and without the hepatopancreas intact; however, the cooking water contained 80% of the PCBs lost from the crab. NJDEP (2006c) reports that there is no specific cooking method available to reduce the chemical contaminant levels in Blue crabs. Because the crab is cooked whole, even if the consumer does not eat the hepatopancreas, exposure to the chemical contaminant may still potentially occur if the crab is cooked before the hepatopancreas is removed and if the liquid used to boil the crab is used in juices, sauces, bisques, or soups. It is assumed for this evaluation that the cooking liquid is consumed along with the crab meat. Therefore, cooking loss for crabs is assumed to be 0% for the RME and CTE because data are not currently available from USEPA or published literature to support any type of reduction in concentrations under this type of exposure scenario.

Table 5-6. Range of Cooking Losses from Fish.[a]

COPC	Exposure Scenario	
	RME (%)	CTE (%)
DDD	0	30[b]
DDE	0	35[b]
DDT	0	30[b]
Chlordane	0	33[b]
Dieldrin	0	30[b]
Dioxins	0	49[b]
PCBs	0	20
Mercury[c]	0	0

RME – reasonable maximum exposure

CTE – central tendency estimate

a. Refer to Table 5-5, "combined column".

b. The USEPA EFH (1997) provides a recommended default adjustment for cooking and preparation loss. The values given in the EFH for fish are 30% for Mean Net Cooking Loss and 11% for Mean Net Post Cooking Loss.

c. Preparation and cooking loss adjustments should not be applied for metals in most cases (USEPA, 2000a).

Exposure Frequency (EF)

The ingestion rates for fish and crabs are annualized and presented on a daily basis. Therefore, the exposure frequency for the fish and crab consumption is assumed to be 365 days per year (USEPA, 1989) for both the RME and CTE scenarios.

Exposure Duration (ED)

For the adult angler/sportsman, exposure is assumed to occur for six years as a child and 24 years as an adult, for a total RME exposure duration of 30 years. An exposure duration of nine years is assumed for the CTE. These assumptions are based on recommendations by USEPA (1989 and 1991) and represent upper bound and average residential tenure at a single location. For the angler/sportsman adolescent, exposure is assumed to occur for nine years (from ages 10 through 18 years) for the RME, and for the CTE exposures the residential default of six years is used (USEPA, 1991). The residential default of six years for the RME is assumed for the child receptor (USEPA, 1991) and the RME is halved for the CTE.

Connelly *et al.* (1992) found that individuals may travel up to 37 miles to fish. The potential exists that individuals may live in one section of the 7-mile stretch of the Passaic River and travel to another portion of the river to fish or crab. Therefore, the potential exists that individuals may be exposed for longer periods of time than the 30 years identified in this evaluation. During the RI/FS, in and out migration census data will be evaluated to determine if the ED is longer than 30 years. The use of the 30 year ED may potentially underestimate the cancer risks for this site.

Body Weight (BW)

Age-specific body weights are used in this evaluation. For the adult and child receptors, the default weights of 70 kg and 15 kg are used (USEPA, 1991). For the adolescent receptor, the applicable weight of 54.5 kg is used which was derived by averaging the mean body weight estimates for males and females age 10 years to 17 years (USEPA, 2002b). Although the adolescent receptor evaluated in this HHRE is assumed to be from 10 to 18 years of age, the recommendation provided in USEPA (2002b) is to use the male and female mean body weight estimates based on data from the third National Health and Nutrition Examination Survey (NHANES III) summarized in Table 11-6 of the USEPA guidance, which only

presents data up to 17 years of age. Body weight estimates from NHANES II also were provided in USEPA (2002b) which presented male and female mean body weight data up to 18 years of age. Using these data, an estimated mean adolescent body weight (for males and females aged 10 to 18 combined) is 53.1 kg, slightly lower than the 54.5 kg derived using the recommended NHANES III body weight values. According to USEPA (2002b), an upward trend in body weight was observed between NHANES II (1976-1980) to NHANES III (1988-1994) which still may be valid. Given the upward trend in body weights over the years, and the USEPA recommendation to use data from NHANES III, the body weight of 54.5 kg is used for the adolescent receptor.

5.2 Toxicity Assessment

The toxicity assessment determines the relationship between the magnitude of exposure to a COPC and the nature and magnitude of adverse health effects that may result from such exposure. For purposes of this evaluation, COPCs are classified into two broad categories: noncarcinogens and carcinogens. Toxicity studies with laboratory animals or epidemiological studies of human populations provide the data used to develop toxicity criteria.

Carcinogens are agents that induce cancer. Potential carcinogenic effects are expressed as the probability that an individual will develop cancer from a lifetime based on the exposure assumptions used in the risk assessment. The cancer slope factor (CSF) is a plausible upper bound estimate of carcinogenic potency used to calculate cancer risk from exposure to carcinogens, by relating estimates of lifetime average chemical intake to the incremental probability of an individual developing cancer over a lifetime. CSFs are derived based on an analysis of the animal and/or human data to determine the most appropriate model to use in the extrapolation from animal to humans or direct use of human epidemiological studies (USEPA, 1996; 1999; 2005b). Chemical specific CSFs use data to determine whether a threshold exists or if the chemical is a non-threshold carcinogen (USEPA, 2005b). The slope factor is protective and assumes that exposure to any concentration of a carcinogen has the potential to produce an increased risk. The CSFs developed by the USEPA are plausible upper bound estimates, which means that the USEPA is reasonably confident that the actual cancer risk will not exceed the estimated risk calculated using the CSF. Cancer risks from exposure to multiple carcinogens and multiple pathways are assumed to be additive (USEPA, 1989; 2000b).

Noncarcinogenic health effects were evaluated using reference doses (RfD) developed by USEPA. A RfD is an estimate of a daily oral exposure for a given duration to the human population (including susceptible subgroups) that is likely to be without an appreciable risk of adverse health effects over a lifetime [USEPA's Integrated Risk Information System (IRIS) definition]. RfDs are expressed in milligrams of contaminant per kilogram of body weight per day (mg/kg-day). The RfD is a health-based criterion based on the assumption that thresholds exist for non-cancer health effects (*e.g.*, liver or kidney damage) over a length of time of exposure (*e.g.*, chronic). Chronic RfDs are specifically developed to be protective against long-term exposure to a contaminant.

A table summarizing the toxicity criteria, target organ, weight of evidence classifications, uncertainty factors, and other relevant information for each chemical is provided in Attachment C, Tables C-11 and C-12 for non-cancer and cancer toxicity, respectively. Toxicity criteria have been selected according to the USEPA (2003) OSWER Directive 9285.7-53 that recommends a hierarchy of human health toxicity values for use in risk assessments at Superfund sites. The hierarchy is as follows: 1) USEPA's IRIS; 2) USEPA's (Office of Research and Development, National Center for Environmental Assessment, Superfund Health Risk Technical Support Center) Provisional Peer-Reviewed Toxicity Values (PPRTVs), and 3) other sources of information, such as toxicity values from the State of California's Environmental Protection Agency (CalEPA) and the ATSDR minimal risk levels (MRLs) for noncarcinogenic constituents.

At the current time, USEPA is reassessing the toxicity of dioxins and related compounds. In 2006, the National Academies of Science evaluated USEPA's 2003 dioxin reassessment and provided comments which are currently being reviewed by USEPA. This evaluation used the toxicity values available in Health Effects Assessment Summary Tables (HEAST) for dioxin as the basis for the cancer risk evaluation for the dioxin; non-cancer health effects were evaluated qualitatively. The RI/FS that will be developed in the future will evaluate the status of USEPA's reassessment to determine whether modifications are necessary. In July 2006, the World Health Organization (WHO) released its re-evaluation of human and mammalian TEFs for dioxins and dioxin-like compounds performed in 2005. The HHRE was completed using the 1998 TEFs. Dioxin/furan and PCB congeners with revised TEFs are summarized below in Table 5-7. Calculations for this HHRE were performed using the WHO 1998 TEFs. For this risk evaluation, TEQs may have been underestimated or overestimated based on the revised TEFs as further discussed in the Uncertainty Analysis (Section 5.4).

Table 5-7. Dioxin/furan and PCB Congeners with Updated TEFs.

Congener	WHO 1998 TEF[a]	WHO 2005 TEF[b]
Chlorinated dibenzo-p-dioxins		
OCDD	0.0001	**0.0003**
Chlorinated dibenzofurans		
1,2,3,7,8-PeCDF	0.05	**0.03**
2,3,4,7,8-PeCDF	0.5	**0.3**
OCDF	0.0001	**0.0003**
Non-ortho substituted PCBs		
PCB 81	0.0001	**0.0003**
PCB 169	0.01	**0.03**
Mono-ortho substituted PCBs		
PCB 105	0.0001	**0.00003**
PCB 114	0.0005	**0.00003**
PCB 118	0.0001	**0.00003**
PCB 123	0.0001	**0.00003**
PCB 156	0.0005	**0.00003**
PCB 157	0.0005	**0.00003**
PCB 167	0.00001	**0.00003**
PCB 189	0.0001	**0.00003**

a. Source: Van den Berg *et al.,* 1998.
b. Source: Van den Berg *et al.,* 2005.

Commercial PCBs tested in laboratory animals were not subject to prior selective retention of persistent congeners through the food chain (*i.e.*, laboratory test animals were fed Aroclor mixtures, not environmental mixtures that had been bioaccumulated). According to USEPA's analysis of published studies, bioaccumulated PCBs appear to be more toxic than commercial PCBs and appear to be more persistent in the body (USEPA, 1996; 1999). CSFs of 2.0 and 1.0 $(mg/kg\text{-}day)^{-1}$ are used to evaluate cancer risks for the upper-bound and central estimate exposures to PCBs *via* ingestion of fish from the Passaic River (Table C-12). The CSFs are based on the IRIS chemical file which is based on the 1996 PCB reassessment (USEPA, 1996). Two RfDs are available for PCBs, one for Aroclor 1016 and the

other for Aroclor 1254. For the non-cancer toxicity assessment, the RfD for Aroclor 1254 is used to assess non-cancer toxicity since the bioaccumulation of PCBs is more consistent with the more heavily chlorinated Aroclor 1254. Dioxin-like PCBs also have been evaluated. TEFs for these congeners are summarized in Table 5-7.

All other chemicals were evaluated using the toxicity values presented in their respective IRIS chemicals files.

5.3 Risk Characterization

Risk characterization involves an estimation of the magnitude of the potential adverse health effects associated with the COPCs. It also includes summary judgments about the nature of the human health threat to the defined receptor populations. The risk characterization combines the results of the dose-response (toxicity assessment) and exposure assessment to calculate cancer risks and non-cancer health hazards. In accordance with USEPA's guidelines for evaluating the potential toxicity of complex mixtures (USEPA, 1986; 2000b), this assessment assumes that the effects of all constituents are additive through a specific pathway within an exposure scenario (USEPA, 1986; 2000b).

Risks are estimated as probabilities for COPCs that elicit a carcinogenic response. The excess lifetime cancer risk is the incremental increase in the probability of developing cancer associated with exposures to contaminated media at the site. A risk of 1×10^{-6} for example, represents the probability that one person in one million persons exposed to a carcinogen over a lifetime (70 years) will develop cancer. The upper-bound excess lifetime cancer risks derived in this assessment are compared to the regulation of the NCP that includes a risk range of 10^{-4} (one in ten thousand) to 10^{-6} (one in a million) (USEPA, 1990).

The excess cancer risk is estimated using CSFs where risk is directly related to intake (USEPA, 1989):

$$\text{Risk} = \text{CSF} \times \text{LADD} \qquad (5\text{–}7)$$

where:

Risk	=	Excess lifetime cancer risk (probability)
CSF	=	Cancer Slope Factor $(\text{mg/kg-day})^{-1}$
LADD	=	Lifetime average daily dose (mg/kg-day)

Only LADDs are used in conjunction with CSFs to obtain excess lifetime cancer risk estimates because slope factors are based on average lifetime exposures. CSFs are derived for specific routes of exposure and, because the primary route of exposure to humans is ingestion, only oral toxicity values will be applied in this evaluation. Cancer risks from exposure to multiple carcinogens will be assumed to be additive (USEPA, 1989). To estimate the total excess cancer risks from all carcinogens, cancer risks from each compound will be summed. Excess cancer risks that are less than the acceptable NCP risk range will be identified as *de minimis* risk.

The potential for noncarcinogenic health effects is estimated by comparing the average daily dose (ADD) of a compound with the RfD based on the specific route of exposure (***e.g.,*** oral). The ratio of the intake to reference dose (ADD/RfD) for an individual chemical is termed the hazard quotient (HQ). An HQ greater than 1 indicates the potential for adverse health effects, as the RfD is exceeded by the intake (USEPA, 1986). These ratios are calculated for each chemical that elicits a noncarcinogenic health effect when a RfD is available for the chemical. HQs less than 1 indicate that no adverse health effects are predicted from exposure to COPCs. An HQ greater than 1 indicates that exposure to that contaminant may cause adverse health effects in exposed populations. It is important to note, however, that the Hazard Index (HI) exceeding 1 does not predict a specific disease.

Typically, chemical-specific HQs are summed to calculate pathway HI values. The HI is calculated by summing all HQs for all noncarcinogenic constituents through an exposure pathway:

$$HI = HQ_1 + HQ_2 + \ldots + HQ_j = (ADD_1/RfD_1) + (ADD_2/RfD_2) + \ldots + (ADD_j/RfD_j) \quad (5\text{–}8)$$

where:

HQ_j = Hazard Quotient of the j^{th} chemical
ADD_j = Average Daily Dose of the j^{th} chemical
RfD_j = Reference Dose for the j^{th} chemical

This approach can result in a situation where HI values exceed 1 even though no chemical-specific HQs exceed 1 (*i.e.,* adverse systemic health effects would be expected to occur only if the receptor were exposed to several contaminants simultaneously). In this case, chemicals are segregated by similar effect on a target organ, and a separate HI value for each effect/target organ is calculated (USEPA, 1989). If any of the separate HI values exceed 1.0, adverse, noncarcinogenic health effects are possible.

5.3.1 RME Results

The cancer risks associated with current conditions are summarized in Tables C-13 through C-15 for the RME and depicted on Figure 5-8. The calculated total cancer risks for the adult sportsman/angler [estimated for a 30-year exposure duration (ED) by summing the risks for the adult (based on 24-year exposure) and the child (based on 6-year exposure) are 1×10^{-2} and 2×10^{-2} for ingestion of fish and crab, respectively. The ingestion risks for the adolescent receptor are 2×10^{-3} and 4×10^{-3} for fish and crab, respectively. TCDD TEQ (D/F), TCDD TEQ (PCBs), and total PCBs are the primary contributors to a combined risk above 1×10^{-2} for ingestion of both fish and crab, with individual cancer risks above 1×10^{-4} for each receptor, which exceeds the risk range described above. Approximate contributions to total risk from TCDD TEQ (D/F), TCDD TEQ (PCBs), and total PCBs are 65%, 20%, and 10%, respectively. For ingestion of fish, the risk for chlordane was estimated at 1×10^{-4}, contributing approximately 1% to the total risk. However, the estimated risk associated with chordane for ingestion of crab was much lower at 2×10^{-6}. TCDD TEQ (D/F) comprises over three quarters of the risk associated with the dioxins [*i.e.*, TCDD TEQ (D/F) and TCDD TEQ (PCBs)]. As shown on Figure 5-8, RME cancer risks are outside the risk range of 1×10^{-4} and 1×10^{-6} (USEPA, 1990). These risks are the risks associated with continuance of current EPCs throughout the exposure duration. The future risks under a No Action alternative may need to account for any changes in the EPCs over time, this is discussed further in Section 8.1.1.

The non-cancer HIs are summarized in Tables C-16 through C-18 and shown on Figure 5-9. HIs for the adult, adolescent, and child are 64, 55, and 99 for ingestion of fish, respectively. For ingestion of crab, the HIs are 86, 72, and 140 for the adult, adolescent, and child receptors. Total PCBs are the primary contributor to the excess hazard for all receptors for both ingestion of fish and crab. The HQ for ingestion of methyl mercury in fish is 1 for the adult and adolescent receptors and slightly higher at 2 for the child receptor. In addition, the HQs for ingestion of chlordane in fish for all of the receptors are greater than 1. Exceedence of the NCP criterion of 1 is clearly indicated for the fish and crab RME scenarios as depicted on Figure 5-9.

5.3.2 CTE Results

The cancer risks are summarized in Tables C-19 through C-21 for the CTE and depicted in Figure 5-8. The calculated total cancer risks for the 30-year exposure duration (*i.e.*, angler/sportsman adult + child

receptors) are 3×10^{-4} and 1×10^{-3} for ingestion of fish and crab, respectively. The ingestion risks for the adolescent receptor are 6×10^{-4} and 4×10^{-3} for ingestion of fish and crab, respectively. The ingestion risks for the adolescent receptor are 2×10^{-4} and 2×10^{-3} for fish and crab, respectively. TCDD TEQ (D/F) and TCDD TEQ (PCBs) in fish and TCDD TEQ (D/F) and TCDD TEQ (PCBs) and total PCBs in crab are the primary contributors to the total cancer risks. Only the individual cancer risks for fish for TCDD TEQ (D/F) exceeds 1×10^{-4}. However, the individual cancer risks for crab for TCDD TEQ (D/F), TCDD TEQ (PCBs), and total PCBs are at or above 1×10^{-4} for each receptor. Estimated risks for chlordane were much lower at 9×10^{-6} and 6×10^{-7} for fish and crab ingestion, respectively. As shown on Figure 5-8, CTE cancer risks are above the risk range of 1×10^{-4}.

The non-cancer HIs are summarized in Tables C-22 through C-24 and shown on Figure 5-9. HIs for the adult, adolescent, and child are 16, 14, and 25 for ingestion of fish, respectively. For ingestion of crab, the HIs are 60, 53, and 87 for the adult, adolescent, and child receptors. Total PCBs are the primary contributor to the excess hazard for all receptors for both ingestion of fish and crab. The HQ for ingestion of methyl mercury in fish is less than 1 for each of the receptors. Exceedence of the NCP criterion of 1 is clearly indicated for the fish and crab CTE scenarios as depicted on Figure 5-9.

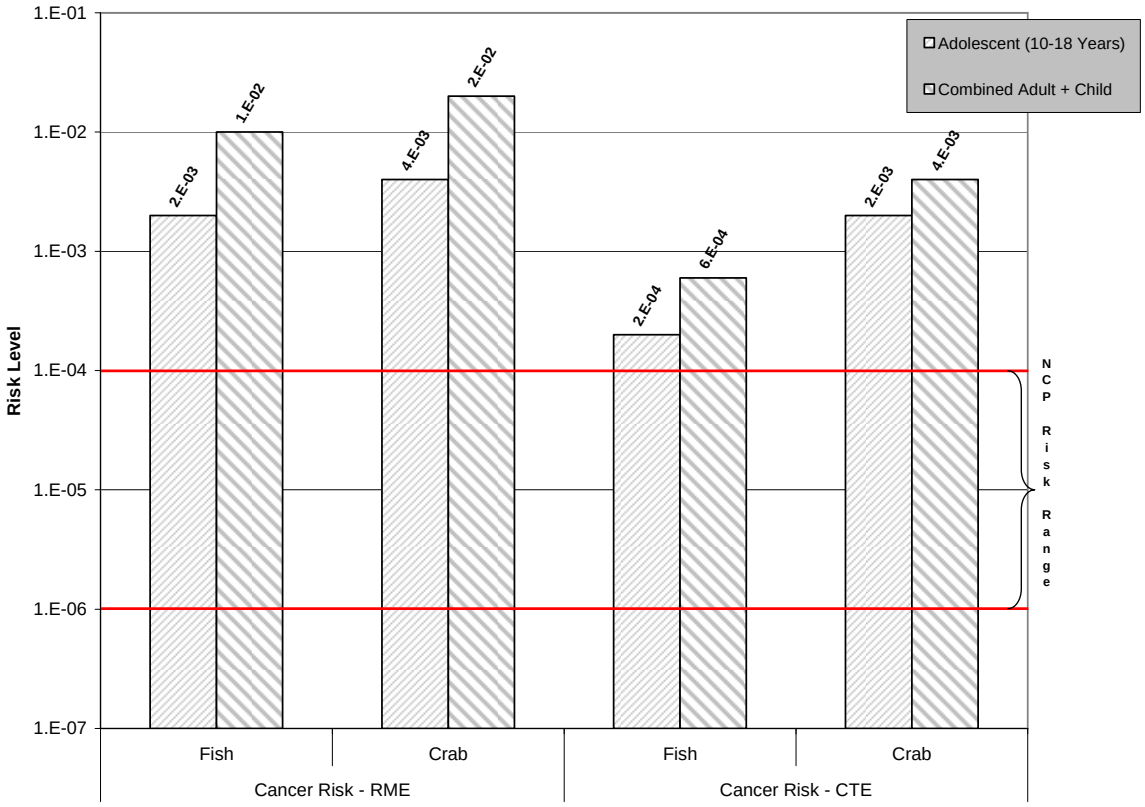


Figure 5-8. Current Cancer Risks for RME and CTE.

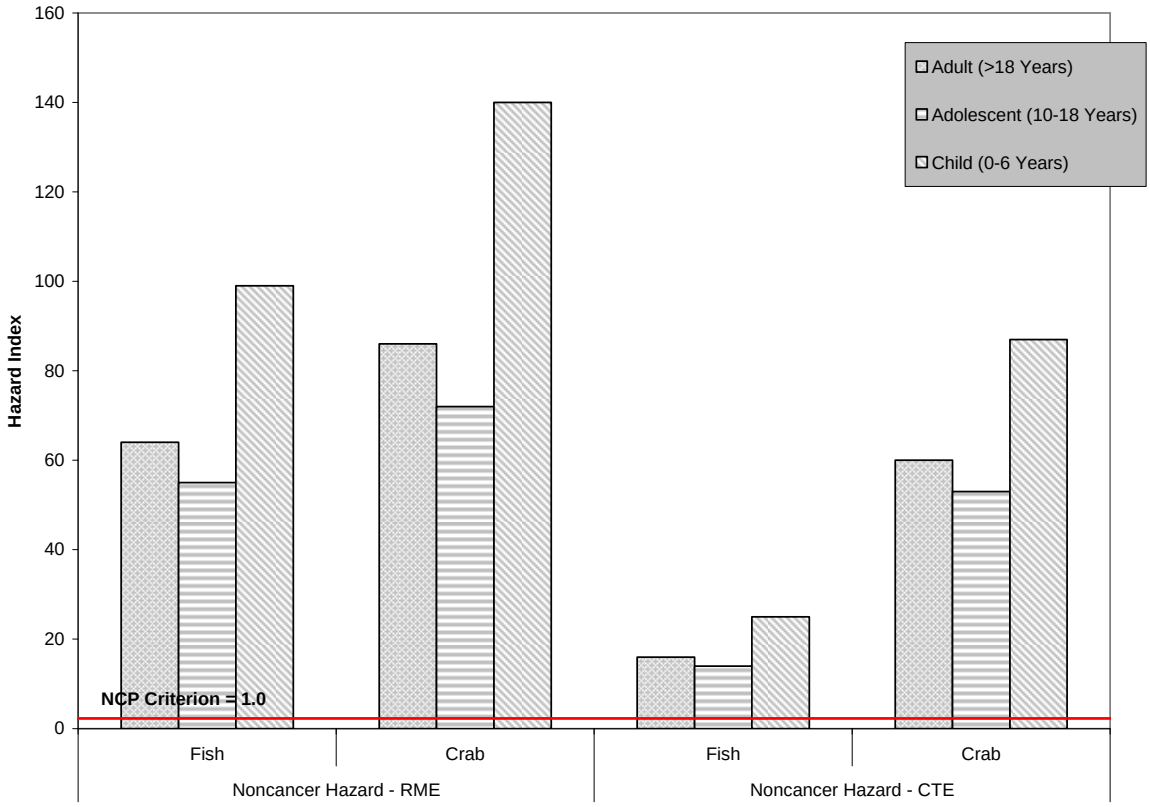


Figure 5-9. Current Non-cancer Hazards for RME and CTE.

5.4 Human Health Uncertainty Analysis

This risk evaluation is consistent with Agency guidance, guidelines and policies. The application of these procedures is designed to reduce potential uncertainty and assure consistency.

A qualitative evaluation is provided in this section to address uncertainties associated with the estimates of risk/hazard that have been presented in this report. Risk results presented in this report are best estimates based on the most recent information and techniques available for predicting risk. Two primary sources of uncertainty associated with risk estimates are:

- Model uncertainty (*i.e.,* methods/models used to calculate EPCs and risk); and,
- Parameter uncertainty (*i.e.,* uncertainty in model input parameter exposure variables).

For the evaluation of risk in response to existing concentrations, model uncertainty is not discussed because standard, accepted exposure and risk models have been employed in this evaluation; therefore, it is assumed that the formulations of the models used to predict exposure and risk are valid at this time. Large uncertainties can often arise in risk estimates that are based on models that simulate the fate/transport of contaminants. However, risks here are based on measured contaminant data and there is no dependency on the use of fate/transport modeling to predict EPCs for current conditions. However, uncertainty in the projection of future exposure concentrations is a major source of uncertainty in the estimate of LADD and associated cancer risks under the no action alternative presented in Section 8.0.

Conversely, parameter uncertainty is discussed here because this type of uncertainty is the most likely source of uncertainty impacting the calculated cancer risks and non-cancer health hazards. Parameters involved in the risk evaluation are categorized according to the step in which they occur (*i.e.,* hazard identification, exposure assessment, dose-response [toxicity] assessment, and risk characterization). The various parameter uncertainties and the likely impact of these uncertainties on the calculated risks are summarized in this section and in Table 5-8.

The following discussion identifies uncertainties based on over or underestimates of cancer risks and non-cancer health hazards.

One of the major uncertainties associated with the hazard identification process is the identification of COPCs. Not all of the COPCs identified in biota were evaluated. Only a subset of contaminants that capture the chemicals with the greatest potential to bioaccumulate through the food chain and the primary risk drivers were carried through the risk evaluation. In addition, COPCs associated with other environmental media (*e.g*., sediment and surface water) in conjunction with other potentially complete exposure pathways (*e.g*., dermal contact, incidental ingestion) were not included in the risk evaluation because the ingestion of biota and the COPCs identified for this medium are thought to drive risks and therefore cleanup objectives. In the absence of the quantification of these additional risk pathways and COPCs the risks may be underestimated.

There is a concern for the potential to double count PCB concentrations and PCB risk when both dioxin-like PCB congener data and total PCB (as Aroclor) data are used to determine risk, and with those risks then being added together. Therefore, select PCB data were reviewed to address this concern. The results of the PCB enhancement assessment is presented in Attachment C. Briefly, the results of the assessment indicate that the total PCB concentration (and total PCB-based risk) would be reduced by 8% if the contribution of the 12 dioxin-like PCB congeners is considered (*i.e.*, subtracted from the total PCB concentration). The overall risk associated with PCBs therefore would be reduced by 1% to 3%, resulting in the total cancer risk value declining by approximately 1% for both fish and crab risk estimations. This decrease in the total cancer risk, however, does not significantly impact the risk values. The estimated risk values at one significant figure would still be reported as the same values summarized in Section 5.3 and presented in Attachment C.

Although methyl mercury was identified as a COPC, tissue data for methyl mercury was fairly sparse. As a result, analytical data for total mercury were used to represent methyl mercury results. This assumes that all the mercury bioaccumulated in the food chain is present as methyl mercury in the tissue, which is generally a reasonable assumption for human exposure via ingestion of piscivorous fish; however, this assumption may result in an overestimate of the non-cancer health hazards.

Several parameters associated with the exposure assessment have uncertainties associated with them that impart uncertainty to the calculated cancer risks and the non-cancer health hazards. These include EPCs, potential receptors, and exposure assumptions evaluated. Each of these is discussed below.

- Based on USEPA risk assessment guidance (USEPA, 1989), the 95% UCL of the arithmetic mean is used as the EPC because it is a health protective estimate of the average site-wide concentration that a receptor would be exposed to. The UCL is a statistic and thus by nature is uncertain; however, to minimize the uncertainty in the EPCs, UCLs were calculated using several statistical methods and the most appropriate value was selected based on factors such as distribution of the raw data (*e.g.*, normal, lognormal). The UCL is used to represent the reasonable maximum exposure encountered at the site; therefore, risks may be overestimated for some receptors who may experience less than the reasonably maximum exposures.

- For non-detected values, one-half the detection limit is assumed (USEPA, 1989). Risks for some compounds with low frequency of detection may be overestimated by this approach. Potentially larger errors may be present in the sum of Aroclors used to estimate hazard associated with PCBs.
- Historical data (from 1994 to 2001) used to calculate the EPC for fish included samples consisting of whole body, rather than fillet. Incorporation of all portions of the fish may result in overestimating the concentrations if in fact individuals tend to eat mainly fillets. The calculated risks may overestimate the cancer risks and non-cancer health hazards.
- Information regarding the specific characteristics of crab and fish samples was missing from the database. This includes data that would correlate with the age, length, weight, and sex of the fish. This lack of information could be a potential cause of uncertainty in the estimation of exposure concentrations and whether the fish/crab was of a consumable size for human consumption.
- Similarly, historical data used to calculate the EPCs for crab incorporated the hepatopancreas for results labeled as "edible tissue". Based on the biology of the crab, the potential exists for the hepatopancreas to be ingested while eating the other tissues from the crab. In addition, several cultures specifically consume the hepatopancreas as a delicacy. Incorporation of this organ results in an overestimation of the EPC concentration, resulting in an overestimate of risk.
- Use of a the white perch and American eel to derive the EPC for fish ingestion, assumes individuals consume only white perch and American eel from the Lower Passaic River and that each of these species is equally consumed adds uncertainty to the risk estimate. Risk estimates for individuals who only consume white perch would be underestimated because concentrations in white perch were always higher than the American eel. Averaging the two fish species would therefore dilute the EPC. On the other hand, the risk for those individuals consuming only American eel would be overestimated. This may result in an underestimation or overestimation of risks.
- There is uncertainty in the receptors that were evaluated in the risk evaluation and their angling activities/habits. To minimize uncertainty in the calculated risks, exposure assumptions and parameters for these receptors were obtained from published literature sources (*e.g.,* creel surveys) for the Lower Passaic River or surrounding areas. In some instances, exposure assumptions and parameters were based on professional judgment and default exposure values recommended by USEPA. Risks are more likely to be overestimated than underestimated because of the conservative nature of the exposure assumptions. The uncertainties in the calculated risks are high. The possibility exists for subsistence populations who live in the area may consume higher amounts of fish/crab than a recreational angler. Crab consumption was assessed based on a Creel Survey of the Newark Bay Complex which includes the Passaic River study area. However, it was noted that specific distributions of fish ingestion were not available for the survey. The potential exists that the risks may be either underestimated or overestimated.
- Angling, crabbing, and consumption of catch within the lower portion of the Lower Passaic River was assumed to be a frequent event for the receptors even though this portion of the river is industrial in nature and fish and crab advisories are in existence. However, there is evidence that individuals do fish in this area, but the time spent and the amount caught is uncertain. There is also uncertainty about how changes in water quality might cause changes in the fishing activities on the river over time. As such, the uncertainty in the calculated risks may result in either an underestimation or overestimation of risk.
- The ingestion rate for crab consumption was based on a 3-month period that individuals reported they crab. This rate did not take into consideration the number of meals eaten throughout the remainder of the year when anglers may continue to crab or that the anglers freeze their catch. The ingestion rate may underestimate the risks. Therefore, risks may be somewhat underestimated.

- Exposure to dioxin, dioxin-like compounds, and other bioaccumulative compounds in sensitive subpopulations, such as breast-fed children, was not evaluated quantitatively. These compounds are lipophilic and concentrate in breast milk. Therefore, risks are more likely to be underestimated for these sensitive populations.

As discussed below, the primary aspects of the toxicity assessment that impart uncertainty to the calculated risks include uncertainty in the toxicity data for constituents detected at the site.

- The toxicity assessments included human epidemiological studies in addition to animal studies. Following careful review of the data, the most appropriate studies were used in the development of toxicity values. The toxicity values for dioxins, mercury and PCBs, the primary chemical risk drivers, were extensively peer-reviewed. The toxicity values are designed to be protective of human health and the potential exists that the risks may be lower (USEPA, 2005b).
- The cancer toxicity for dioxins/furans is being evaluated through the USEPA reassessment. Only a cancer assessment was evaluated in this HHRE based the availability of a CSF. The non-cancer assessment or a Margin of Exposure was not calculated. The potential exists that the dioxin risks may be either overestimated or underestimated.
- In July 2006, the WHO released its re-evaluation of human and mammalian TEFs for dioxins and dioxin-like compounds performed in 2005. The HHRE was completed using the 1998 TEFs. Dioxin/furan and PCB congeners with revised TEFs are summarized in Table 5-7. In the 2006 WHO re-evaluation, some of the chlorinated dioxin/furan TEFs increased by factors ranging from 1.7 up to 3 over the corresponding 1998 values. Others decreased by a factor of 1.7. The non-ortho substituted PCBs all increased by a factor of 3, whereas the mono-ortho substituted PCBs decreased by factors ranging from 3.3 to 16.7. Calculations were performed (although not presented in this HHRE) using the revised TEFs and the risk results were virtually unchanged compared to the calculations derived using the 1998 TEF values. For this risk evaluation, TEQs may have been underestimated or overestimated based on the revised TEFs.

Finally, uncertainty in the calculated risks can arise from uncertainty in the way in which risks were calculated or aggregated, as discussed below. Table 5-8 summarizes all of the uncertainties discussed in this section.

- The assessment did not evaluate the potential cancer risks and non-cancer health hazards based on background concentrations. The contributions from background concentrations will be evaluated in the RI/FS. The effect of including background and ambient constituents in the risk assessment is that the calculated risks overestimate the risk that is due to chemical releases from the site.
- Risks were derived assuming that the receptors ate fish or crab, but not both. Although Burger (2002) reported that survey results indicated that the majority of people either fished or crabbed, it is likely that some anglers may eat both fish and crab. Therefore, risks may be underestimated for individuals who eat both fish and crab. However, for individuals eating both crabs and fish at each meal, the respective ingestion rates for both would be expected to decrease (*i.e.,* if someone eats both fish and crabs during a meal, than the fish ingestion rate and the crab ingestion rate may be lower than the respective ingestion rates when only fish or only crab is consumed during a meal). Therefore risks would be overestimated if the same respective consumption rates were assumed for an individual consuming both fish and crabs during a meal. As such, the uncertainties in the calculated risks for this site are considered low.

- The HQ for ingestion of methyl mercury in fish is 1 for the adult and adolescent receptors and 2 for the child receptor based on an EPC of 0.3 mg/kg. The oral RfD for mercury is based on human epidemiological studies and therefore the overall confidence in the RfD for methyl mercury is high. Because the HQ for the child receptor is above 1, there may be concern for potential health effects as a result of methyl mercury exposure. Thresholds which have been used for establishing consumption advisories are 1.0 mg/kg wet weight (used by the Food and Drug Administration [FDA] for restriction of commercial sale of fish) and 0.5 mg/kg (with advisories of no or restricted consumption of fish with higher assessment of total mercury concentrations in fish from rivers, lakes and reservoirs in New Jersey concentrations) (Horwitz *et al*., 2002). In 1994, NJDEP and the Toxics in Biota Committee derived a risk-based criteria for mercury concentrations as low as 0.08 mg/kg as a trigger for state advisories restricting consumption among the most vulnerable segments of the human population (*e.g*., children and pregnant women) (Horwitz *et al*., 2002). The uncertainties associated with the exposure assumptions used in the calculation of the non-cancer HQ for mercury are similar to the other fish contaminants of concern identified above. The information presented regarding the concentration of mercury in fish used to establish fish advisories for the general and vulnerable portions of the human population (*e.g*., children and pregnant women) also identify potential concerns for the ingestion of mercury contaminated fish at varying concentrations.

Table 5-8. Summary of Major Uncertainties in the Human Health Risk Evaluation and Estimated Impacts on Calculated Risks.

Risk Evaluation Step	Source of Parameter Uncertainty	Description of Uncertainty	Impact on Calculated Risks
Hazard Identification	Identification of COPCs for quantitative evaluation	Only a subset of contaminants that capture the primary risk drivers were carried through the risk evaluation process.	Risks are underestimated.
		COPCs associated with other environmental media (*e.g.*, sediment and surface water) were not evaluated.	Risks are underestimated.
	Mercury and Methyl mercury	Due to lack of methyl mercury data in the biota tissue data, results for mercury were used as surrogate for methyl mercury based on fate and transport properties of mercury in the environment and the toxicokinetics of mercury in the biota. This assumes that all mercury contained in fish and crabs eaten by humans is present as methyl mercury.	Risks are likely overestimated.
Exposure Assessment	EPCs for biota	95% UCLs on the mean were calculated from measured data collected from numerous samples distributed across the exposure area and used as the EPC to calculate risk. The difference between the UCL and mean indicates the level of uncertainty associated with EPC estimation.	Risks for some compounds with low frequency of detection may be overestimated by using ½ the detection limit for non-detected values.
	Fish and crab tissue data used to derive EPC	Historical data used to calculate the EPC for fish may have at times included samples consisting of whole body, rather than only fillet. Similarly, historical data used to calculate the EPC for crab incorporated the hepatopancreas results.	Incorporation of all portions of the fish/crab may result in overestimating the concentrations if in fact individuals tend to mainly eat fillets or muscle tissue. Risks for ingestion of crab may have been over estimated because data from the hepatopancreas-specific samples were included in the EPC.
	Use of a the white perch and American eel to derive the EPC for fish ingestion	Use of a weighted average fish concentration, consisting of white perch and American eel, was used to represent a broad range of fish species that could be caught and consumed. However, the assumption is that fish species are equally caught and consumed.	Risks may be over- or underestimated for individuals who only consume a specific species. For example, risks for individuals who only consumed white perch would be underestimated because concentrations in white perch were always higher than the American eel. A weighted average of the two fish species lowered the EPC. On the other hand, the risk for those individuals consuming only American eel would be overestimated.

Table 5–8. Summary of Major Uncertainties in the Human Health Risk Evaluation and Estimated Impacts on Calculated Risks, continued.

Risk Evaluation Step	Source of Parameter Uncertainty	Description of Uncertainty	Impact on Calculated Risks
	Receptors and exposure parameters	Selecting the most representative exposure parameters for the angling activities/habits is difficult, especially for exposure duration, exposure frequency, and fish ingestion rates.	Risks may be over- or underestimated for this site.
	Receptors and exposure parameters	Ingestion rate for consumption of crab was based on a three month period that individuals reported they crab.	This rate did not take into consideration the number of meals eaten throughout the year due to extra crabbing or because crabs were caught and placed in the freezer. Therefore, risks may be underestimated.
		Other potentially complete exposure pathways for the anglers were not included (*e.g.*, dermal contact with sediment). In addition, exposure to dioxin and dioxin-like compounds in sensitive subpopulations such as breast-fed children was not evaluated.	Exclusion of these additional pathways would underestimate the risks for the site.
Toxicity Assessment	Toxicity data (general)	Toxicity values for dioxin, PCBs, and mercury are based on an assessment of animal and human data. In some cases, animal data were used as the basis for the toxicity values that were further extrapolated to humans.	Because the most conservative values available are typically used, risks are more likely to be overestimated than underestimated.
	1998 vs. 2005 TEF values	The WHO released their re-evaluation of human and mammalian TEFs for dioxins and dioxin-like compounds performed in 2005.	Risks using the 2005 TEF values were virtually equal to those based on the 1998 values.
	Dioxin Reassessment	USEPA is conducting a scientific reassessment of the health risks of exposure to dioxin and dioxin-like compounds in light of significant advances in scientific understanding of mechanisms of dioxin toxicity, significant new studies of dioxin's carcinogenic potential in humans and increased evidence of other adverse health effects.	Future modifications for determining cancer and non-cancer effects may lead to an over- or underestimation of risks and non-cancer health hazards.
Risk Characterization	Distinguishing site-related risks from background and/or ambient risks	Contributions from background conditions were not assessed in the risk assessment based on the lack of information.	The calculated risks may be overestimated, but the extent of this overestimation can not be determined.

Table 5–8. Summary of Major Uncertainties in the Human Health Risk Evaluation and Estimated Impacts on Calculated Risks, continued.

Risk Evaluation Step	Source of Parameter Uncertainty	Description of Uncertainty	Impact on Calculated Risks
	Consumption of both fish and crab	Risks were derived assuming that the receptors ate fish or crab, but not both.	Risks may be underestimated for individuals who eat both fish and crab. However, for individuals eating both crab and fish, the ingestion rates for both these would be expected to decrease; therefore, risks would be overestimated if the same ingestion rates were assumed.
	Thresholds which have been used for establishing consumption advisories	The information presented regarding the concentration of mercury in fish used to establish fish advisories for the general and vulnerable portions of the human population (*e.g.*, children and pregnant women) also identify potential concerns for the ingestion of mercury contaminated fish at varying concentrations.	Noncancer risks may be underestimated for vulnerable portions of the population.

6.0 ECOLOGICAL RISK EVALUATION – CURRENT CONDITIONS

The purpose of the ecological risk evaluation (ERE) is to assess and characterize potential risks to ecological receptors under current conditions in the lower eight miles of the Lower Passaic River. This section presents an evaluation of current risk following USEPA (1998) guidance and includes the exposure assessment, toxicity assessment, risk characterization, and uncertainty analysis. A comparison of current risks to post-remediation risks is presented in Section 8.0.

6.1 Exposure Assessment

The exposure assessment determines the degree of co-occurrence between COPECs and the ecological receptors to be evaluated. To do this, EPCs are calculated for each COPEC over the entire 8-mile stretch of river. These are used to estimate exposures associated with direct contact for non-wildlife receptors (*i.e.,* fish) as well as used in the food web models to estimate daily doses to wildlife receptors.

6.1.1 EPCs

As discussed in Section 4.2.3, EPCs for all media evaluated were calculated as the 95% UGL of the arithmetic means of the available data. The EPCs used in this evaluation are defined in Table 4-1.

6.1.2 Dose Model

The exposure assessment estimates the potential exposure of receptors to COPECs identified at the site. An exposure model incorporating natural history information and species characteristics, such as diet composition, ingestion rates, body weights, and foraging ranges, for each wildlife receptor was developed to evaluate the exposure of the receptor to each COPEC. Equation 6-1 is a dose model that is used to assess daily exposure of COPECs to upper-trophic wildlife receptors (*i.e.,* mink and great blue heron) and to characterize exposure:

$$\text{Dose} = \frac{[(C_{sed} \times IR_{sed}) + (C_{food} \times IR_{food})] \times SUF}{BW} \qquad (6\text{-}1)$$

where,

Dose	=	daily dose resulting from ingestion of sediment and food (mg/kg-d)
C_{sed}	=	concentration of COPEC in surface sediment (mg/kg)
IR_{sed}	=	estimate of receptor's daily ingestion rate of surface sediment (kg/d)
C_{food}	=	concentration of COPEC in food tissue (mg/kg)
IR_{food}	=	estimate of daily ingestion rate of food tissue (kg/d)
SUF	=	site use factor (unitless)
BW	=	body weight (kg)

Because the exposure (and therefore dose) for each receptor is different, the exposure factors used in the dose equation vary slightly based on the receptor being evaluated. The exposure parameters for the mink and great blue heron are summarized in Tables 6-1 and 6-2, respectively.

Table 6-1. Exposure Parameters for the Mink.[a]

Exposure Parameter	Abbreviation	Unit	Value	Source
Body weight	BW	kg	0.6	Mitchell, 1961
Daily ingestion rate of sediment[b]	IR_{sed}	kg/day	0.003	assumption
Daily ingestion rate of fish and crabs[c]	IR_{fish}	kg/day	0.17	USEPA, 1993b
Site Use Factor (max of 1)	SUF	unitless	1	assumption

a. Because of the piscivorous nature of this species, its diet will be considered 80% fish and 20% shellfish (crab).
b. The amount of sediment in its diet is estimated here as 2% and multiplied by the daily ingestion rate.
c. Calculated using regression equation for mammals: IR_{food} (g/day) = 0.235 * BW $^{0.822}$ (g)

Table 6-2. Exposure Parameters for the Great Blue Heron.[a]

Exposure Parameter	Abbreviation	Unit	Value	Source
Body weight	BW	kg	2.2	USEPA, 1993b
Daily ingestion rate of sediment[b]	IR_{sed}	kg/day	0.019	assumption
Daily ingestion rate of fish and crabs[c]	IR_{fish}	kg/day	0.39	Kushlan, 1978
Site Use Factor (max of 1)	SUF	unitless	1	assumption

a. Because of the piscivorous nature of this species, its diet will be considered 85% fish and 15% shellfish (crab).
b. Assume 5% of daily food ingestion rate.
c. Calculated using regression equation for wading birds: log(IR_{food}) (g/day) = 0.966 * log(BW) - 0.64 (g)

6.2 Toxicity Assessment

Chemical- and receptor-specific toxicity reference values (TRV) are compared to the ingestion dose estimates to evaluate the potential effects to wildlife associated with exposure to COPECs in the Passaic River, which results in a HQ (Equation 6-2). In general, an HQ above 1.0 indicates the potential for risk; an HQ below 1 indicates a low potential for risk.

$$HQ = dose/TRV \qquad (6\text{-}2)$$

A TRV is defined as a dose level (based on laboratory toxicological investigations) above which a particular ecologically relevant effect may be expected to occur in an organism following chronic dietary exposure and below which it is reasonably expected that such effects will not occur (USEPA, 2005c). TRV derivation may incorporate uncertainty (or extrapolation) factors (ept, 1996; Chapman *et al.,* 1998) to account for a wide range of limitations, such as interspecies sensitivities. The TRVs presented in this document are considered to be sufficiently conservative, and the use of additional uncertainty factors is not scientifically warranted.

Rather than deriving a single point-estimate associated with specific adverse biological effects, both high and low TRVs are derived for each receptor and each COPEC to reflect the variability of potential risk. The low TRV value consistent with a chronic, no observed adverse effects level (NOAEL). It represents a level at which adverse effects are unlikely to occur, and is used to identify sites posing little or no risk. Conversely, the high TRV is an estimator of potential adverse effects, representing a level at which

adverse effects are more likely to occur, and is consistent with a chronic lowest observed adverse effects level (LOAEL).

Table 6-3 is a summary of the TRVs that were identified for the selected COPECs: copper, lead, mercury, LPAHs, HPAHs, total PCBs, TCDD, DDx, and dieldrin. Generally, two separate wildlife TRVs are developed for each COPEC to characterize risk to the two main categories of wildlife receptors (*i.e.,* birds and mammals).

Table 6-3 TRVs Selected from Available Literature.

COPEC	Test Species	Wildlife Receptors	Low TRV[a] (NOAEL)	High TRV[a] (LOAEL)	Reference
Copper	Chicken	Avifauna	4.1	12	USEPA, 2007
Copper	Pig	Mammals	5.6	9.3	USEPA, 2007
Lead	Chicken	Avifauna	1.6	3.3	USEPA, 2005
Lead	Rat	Mammals	4.7	8.9	USEPA, 2005
Mercury[b]	Mallard	Avifauna	0.0078[c]	0.078	Heinz, 1979
Mercury[b]	Mink	Piscivorous mammals	0.055	0.18	Wobeser *et al.,* 1976a,b
LPAH		Avifauna	-	-	Not necessary
LPAH		Mammals	-	-	Not necessary
HPAH		Avifauna	-	-	Not available
HPAH	Mouse	Mammals	1.0	10	Sample *et al*., 1996
Total PCBs	Chicken	Avifauna	0.10	0.40	Chapman, 2003
Total PCBs	Mink	Mammals	0.080	0.096	Chapman, 2003
Total DDx	Brown pelican	Piscivorous birds	0.0028	0.028	Anderson *et al.,* 1975
Total DDx	Rat	Mammals	0.80	4.0	Fitzhugh, 1948; as cited in Sample *et al.,* 1996
Dieldrin	Mallard	Avifauna	0.071	3.8[c]	Nebeker *et al.*, 1992
Dieldrin	Rat	Mammals	0.015	0.030[c]	Harr *et al.*, 1970
TCDD	Pheasant	Avifauna	1.4×10^{-6}	1.4×10^{-5}	Nosek *et al.,* 1992a,b
TCDD	Mink	Piscivorous mammals	8.0×10^{-8}	2.2×10^{-6}	Tillet *et al.,* 1996

[a.] Units are µg COPEC/g body weight-day
[b.] TRV based on methyl mercury.
[c.] High TRVs are equivalent to the LOAEL from the study that the low TRV (NOAEL) was selected; units in µg COPEC/g body weight-day.

Copper (Cu) is a reddish metal that occurs naturally in rock, water, soil, and sediment. It is an essential element at low levels for all organisms including humans and other animals; but at higher levels, toxic effects can occur. Copper can enter the environment through releases from the mining of copper and other metals, and from factories that make or use copper metal or compounds. Copper can also enter the environment through waste dumps, domestic waste water, and combustion of fossil fuels, wood production, fertilizer production, and natural sources such as dust from soils, volcanoes, and forest fires.

Copper strongly adsorbs to organic matter, carbonates, and clay, which reduces its bioavailability. Copper is highly toxic in aquatic environments and causes adverse effects in fish, invertebrates, and amphibians, with all three groups equally sensitive to chronic toxicity (USEPA, 1993b; Horne and Dunson, 1995). Copper bioconcentrates in various organs in both fish and mollusks (Owen, 1981). Toxic effects in birds include reduced growth rates, lowered egg production, and developmental abnormalities (USEPA Region 5). While mammals are not as sensitive to copper toxicity as aquatic

organisms, toxicity in mammals includes a wide range of animals and effects such as liver cirrhosis, necrosis in kidneys and the brain, gastrointestinal distress, lesions, low blood pressure, and fetal mortality. (ATSDR, 2004a; Kabata-Pendias and Pendias, 1992; Ware, 1983; Vymazal, 1995).

TRVs for copper have been developed by USEPA (2007) and are listed in Table 6-4; these values were used in the wildlife exposure modeling conducted to support the Draft FFS.

Table 6-4. TRVs for Copper from USEPA Eco-SSL Document.

COPEC	Test Species	Wildlife Receptor	Low TRVa	High TRVa	Reference
Copper	**Chicken (Gallus domesticus)**	**Avifauna**	**4.05**	**12.1**	**Ankari et al., 1998**
Copper	**Pig (Sus scrofa)**	**Mammals**	**5.6**	**9.34**	**Allcroft et al., 1961**

Lead (Pb) occurs naturally in the environment; however, most of the elevated levels found throughout the environment come from anthropogenic activities such as mining or factories that make or use lead, lead alloys, or lead compounds. Lead is also released into the air during burning of coal, oil, or waste.

Lead partitions primarily to sediments, but becomes more bioavailable under low pH, low hardness, and low organic matter content (among other factors). It can be bioconcentrated from water, but does not bioaccumulate and tends to decrease with increasing trophic levels in freshwater habitats (Eisler, 1988). Fish exposed to high levels of lead exhibit a wide-range of effects including muscular and neurological degeneration and destruction, growth inhibition, mortality, reproductive problems, and paralysis (Eisler, 1988; USEPA, 1976). Lead also adversely affects invertebrate reproduction.

At elevated levels in plants, lead can cause reduced growth, photosynthesis, mitosis, and water absorption (Eisler, 1988). Lead poisoning in higher organisms primarily affects hematologic and neurologic processes and has been associated with lead shot and organolead compounds, but not with food chain exposure to inorganic lead (other than lead shot, sinkers or paint) (Eisler, 1988). Birds and mammals suffer effects from lead poisoning such as damage to the nervous system, kidneys, liver, sterility, growth inhibition, developmental retardation, and detrimental effects in blood (Eisler, 1988; Amdur et al., 1991). Lead adversely affects reproduction, liver and thyroid function, and the immune system (Eisler, 1988).

TRVs for lead have been developed by USEPA (2007) and are listed in Table 6-5; these values were used in the wildlife exposure modeling conducted to support the Draft FFS.

Table 6-5. TRVs for Lead from USEPA Eco-SSL Document.

COPEC	Test Species	Wildlife Receptors	Low TRV[a]	High TRV[a]	Reference
Lead	Chicken	Avifauna	1.6	3.3	Edens and Garlich, 1983
Lead	Rat	Mammals	4.7	8.9	Kimmel *et al.,* 1980

[a] High TRVs are equivalent to the LOAEL from the study that the low TRV (NOAEL) was selected; units in µg COPEC/g body weight – day.

Methyl mercury is the organic, bioavailable fraction of elemental mercury (Hg), which comes from a variety of environmental sources including mine tailings, gaseous emissions, industrial effluent, and atmospheric deposition. The transformation of inorganic mercury to methyl mercury occurs by anaerobic microorganisms in soils and sediment (ATSDR, 1999), as well as in hypoxic bottom waters. When consumed by aquatic organisms such as fish and shellfish; mercury is not purged or easily metabolized and is capable of bioaccumulating and biomagnifying in successive upper-trophic level organisms who feed on contaminated prey.

Piscivorous mammals and birds who consume sufficient amounts of mercury-contaminated prey show signs of mercury toxicoses, including damage to nervous, excretory, and reproductive systems (RAIS, 1998). Although it exhibits a range of toxic effects in several target tissues (*e.g*., liver, kidney), the primary effects of methyl mercury are on the central nervous system. Methyl mercury readily penetrates the blood/brain barrier producing brain lesions, spinal cord degeneration, and central nervous system dysfunctions (Wolfe *et al.,* 1998).

Symptoms of acute methyl mercury poisoning in birds include reduced food intake, weight loss, weakness in wings and legs, difficult maneuvering, and inability to coordinate muscle movement (Wolfe *et al.,* 1998). Methyl mercury is a potent embryo and nervous system stressor in birds with chronic exposures characterized by symptoms that range from embryo lethality (*i.e.,* reduced egg hatchability), reduced clutch size, eggshell thinning, and aberrant juvenile behavior that may include auditory or visual impairment (Wolfe *et al.,* 1998; Eisler, 1987).

Several long-term feeding studies have been conducted using a variety of bird species including mallards, Black ducks, ring-necked pheasants, Japanese quail, chickens, and Great egrets; the most relevant studies are summarized in Table 6-6. These laboratory studies are consistent with a field study of the common loon in northwestern Ontario (Barr, 1986; as cited in Wolfe *et al.,* 1998), which found that reduction in egg laying and aberrant territorial and nest building behavior occurred when concentration of methyl mercury in the diet exceeded 0.2 to 0.3 μg/g wet weight. There is reasonable consistency in the levels of methyl mercury in the diet associated with the onset of significant reproductive effects in chronically exposed birds. Although Heinz (1974; 1975; 1976a,b; 1979) failed to identify a NOAEL value, the recommended LOAEL (0.078 μg methyl mercury/g-day) was selected because these studies are well-defined and they evaluated the most sensitive endpoints over three generations. Results indicate that piscivorous birds may be as sensitive to the effects of methyl mercury intoxication as are ducks.

Reproductive effects of methyl mercury in mammals include developmental alterations that produce behavioral deficits after birth, impaired fertility, and fetal death. Behavioral effects of low doses of methyl mercury were noted in swimming ability, operant learning, avoidance, maze learning, and development of reflexes. At higher doses, changes in spontaneous activity, visual function, vocalization, and convulsions may occur (Wolfe *et al.,* 1998). Several long-term feeding studies have been conducted using a variety of mammal species including river otter, mink, cat, rat, and laboratory mouse. Table 6-6 also summarizes available long-term laboratory feeding studies for mammals.

Work by Wobeser *et al.*(1976a,b) on long-term feeding studies with mink is the basis for the recommended mammalian wildlife TRV for methyl mercury (NOAEL – 0.055 μg methyl mercury/g-day; LOAEL – 0.18 μg methyl mercury/g-day) because the mink is a receptor of concern at the site and the species is known to be sensitive to mercury. The neurological effects that are the basis for the suggested threshold do not relate directly to the typical ecological endpoint types (*i.e.,* mortality, growth, and reproduction); however, intoxicated animals are likely to be less successful at foraging, predator avoidance, and mating, all of which have population-level significance. The selected rat study suggests that chronic reproductive effects also occur at these low exposure levels.

Table 6-6. Summary of Chronic Feeding Studies with Methyl Mercury.

Species	NOAEL (µg/g-d)	LOAEL (µg/g-d)	Effect	Reference
Birds				
Chicken (*Gallus domesticus*)	-	1.1	Growth inhibition	Fimreite, 1970
Ring-necked pheasant (*Phasianus colchicus*)	-	0.18	Reduced survival, reduced egg production	Fimreite, 1971
Mallard (*Anas platyrhynchos*)	**-**	**0.078**	Reduced number viable eggs, reduced duckling growth, reduced chick survival to day 7	Heinz, 1974, 1975, 1976a,b, 1979
Great egret (*Ardea albus*)	-	0.5 µg/g (food)	Behavioral effects including reduced inclination to forage	Bouton *et al.,* 1999
Black duck (*Anas rubripes*)	-	3 µg/g (food)	Reduced clutch size, egg production, hatchability and duckling survival	Finley and Stendell, 1978
Coturnix (Japanese) quail (*Coturnix japonica*)	8 µg/g food	32 µg/g food	Enzyme induction (AChE, LDH)	Hill and Soares, 1984
Mammals				
Mink (*Mustela vison*)	**0.055**	**0.18**	Anorexia, ataxia; nerve tissue lesions	Wobeser *et al.,* 1976a,b
River otter (L*utra canadensis*)	-	2 µg/g[a]	Anorexia and ataxia	O'Connor and Nielsen, 1981
Cat (*Felis domesticus*)	0.020	0.046	Ataxia, loss of balance, motor incoordination	Charbonneau *et al.,* 1974; 1976
Rat (*Rattus norvegicus*)	0.032	0.16	Reproduction	Verschuuren *et al.,* 1976
Mouse (*Mus musculus*)	0.15	0.73	Sensory neuropathy, cerebral and cerebellar neuronal necrosis	Hirano *et al.,* 1986

Note: Bolded values indicate the selected TRV.
[a] Concentration in diet (wet weight basis).

Polycyclic Aromatic Hydrocarbons (PAHs) are a group of ubiquitous chemicals that are a major component of petroleum products (*i.e*., petrogenic) or are formed during the incomplete burning of coal, oil, gas, wood, garbage, or other organic substances (*i.e.*, pyrogenic). There are more than 100 different PAHs, which generally occur as complex mixtures. Pyrogenically-derived PAHs mainly enter the environment as releases to air from volcanoes, forest fires, residential wood burning, and exhaust from automobiles and trucks; whereas petrogenically-derived PAHs are typically released as direct spills to surface water, soils or sediments. PAHs include some compounds that are highly potent carcinogens that can produce tumors in some organisms at even single doses; but other non-cancer-causing effects are not well understood (Eisler, 1987). Their effects are wide-ranging within an organism and have been found in many types of organisms, including non-human mammals, birds, invertebrates, plants, amphibians, fish, and humans. However, their effects are varied and so generalizations cannot be readily made. Effects on benthic invertebrates include inhibited reproduction, delayed emergence, sediment avoidance, and mortality. Fish exposed to PAHs in sediment and surface water have exhibited fin erosion, liver abnormalities, cataracts, and immune system impairments leading to increased susceptibility to disease (Fabacher *et al*., 1991; Weeks and Warinner, 1984; 1986; O'Conner and Huggett, 1988, Payne *et al.*, 2003). Early mechanistic models categorized effects of individual PAHs as either being receptor mediated (*e.g.,* AhR) with metabolites forming DNA adducts or generally narcotic in nature; however, recent studies suggest that the toxicology is much more complicated (Barron *et al*., 2004; Incardona, *et al*., 2006).

Mammals can absorb PAHs by inhalation, dermal contact, or ingestion (Eisler, 1987). The oral toxicity of PAHs ranges from very toxic to moderately toxic in rats. In addition to tumor induction, other effects

in mammals include adverse effects on reproduction, development, and immunity (ATSDR, 1995). Although a large literature on the effects of oil spills on birds is available, toxicity data for birds associated with the ingestion pathway are limited and no PAH TRV for this receptor group was developed. There are also limited mammalian data available for the 2- and 3-ring PAHs (which are not anticipated to be bioavailable to wildlife at any rate). As a result, only a mammalian TRV for High Weight PAHs was selected to support the Draft FFS. As summarized in Sample *et al.*(1996), Mackenzie and Angevine (1981) exposed female mice via oral intubation during days 7-16 of gestation. The study included three dosing levels and various reproductive endpoints including pregnancy rates, percentage of viable litters, and pup weights were measured. Pup weights were significantly reduced at all dose levels and a LOAEL of 10 µg/g-day was identified; a chronic NOAEL was estimated by applying a 10-fold uncertainty factor (1 µg/g-day) (Sample *et al*., 1996).

Polychlorinated biphenyls (PCBs) are mixtures of up to 209 individual chlorinated compounds (known as congeners). Some commercial PCB mixtures are known in the United States by their industrial trade name, Aroclor. Because they do not burn easily and are good insulating materials, PCBs were used widely as coolants and lubricants in transformers, capacitors, and other electrical equipment. The manufacture of PCBs stopped in the United States in 1977 because there was evidence that PCBs build up in the environment and may cause harmful effects. Once released into the environment, PCBs do not readily break down and therefore may remain for long periods of time, cycling between air, water, and soil. As a consequence, PCBs are ubiquitously found all over the world. The World Health Organization (WHO) has recognized 12 PCB congeners that are structurally similar to dioxins and have similar toxic effects. These congeners are listed in Table 6-7.

PCBs are taken up into the bodies of small organisms and fish in water. They are also ingested by other animals who feed on these aquatic animals. PCBs especially accumulate in fish and marine mammals (such as seals and whales) reaching levels that may be many thousands of times higher than in water.

Animals exposed to PCBs show various kinds of health effects, including anemia, acne-like skin conditions, and liver, stomach, and thyroid gland injuries (ATSDR, 2000). Other effects include reductions in the immune system function, behavioral alterations, and impaired reproduction (ATSDR, 2000). Some PCBs can mimic or block the action of hormones from the thyroid and other endocrine glands. Because hormones influence the normal functioning of many organs, some of the effects of PCBs may result from endocrine changes. Inhalation and dermal exposure to PCBs may cause liver, kidney, and skin damage in animals (ATSDR, 2000).

Table 6-7. 12 Dioxin-like PCB Congeners.

3,3',4,4'-Tetrachlorobiphenyl (PCB 77)
3,4,4',5-Tetrachlorobiphenyl (PCB 81)
3,3',4,4',5-Pentachlorobiphenyl (PCB 126)
3,3',4,4',5,5'-Hexachlorobiphenyl (PCB 169)
2,3,3',4,4'-Pentachlorobiphenyl (PCB 105)
2,3,4,4',5-Pentachlorobiphenyl (PCB 114)
2,3',4,4',5-Pentachlorobiphenyl (PCB 118)
2',3,4,4',5-Pentachlorobiphenyl (PCB 118)
2,3,3',4,4',5-Hexachlorobiphenyl (PCB 156)
2,3,3',4,4',5'-Hexachlorobiphenyl (PCB 156)
2,3',4,4',5,5'-Hexachlorobiphenyl (PCB 167)
2,3,3',4,4',5,5'-Heptachlorobiphenyl (PCB 189)

A TEQ approach will be employed to normalize the evaluation of potential risks associated with wildlife exposure to compounds with dioxin-like toxicological properties (including certain PCB congeners). Consequently, the specific TRVs for PCBs will be used to evaluate the non-dioxin like effects attributable to PCB compounds.

The selected avian TRVs for PCBs are based on an analysis conducted by USEPA Region 5 (Chapman, 2003) for the chicken, which is believed to be one of the most sensitive bird species. These results are summarized in Table 6-8. TRVs were developed individually for Aroclors 1242, 1248, and 1254 based on reported dose response data from multiple collated studies for Aroclor exposure and growth or reproductive effects in chickens. The interpolated no effect and low effect doses for Aroclor 1242 (NOAEL – 0.1 μg PCB/g-day; LOAEL – 0.4 μg PCB/g-day) were selected as the avian TRVs for total PCBs.

Table 6-8. Summary of Avian TRVs for PCB Mixture Based on Chicken Data.

Aroclor Mixture	No Effect[a] (μg/g-d)	Low Effect[a] (μg/g-d)	Effect	Reference
Aroclor 1242[b]	**0.1**	**0.4**	Chick hatchability	Chapman, 2003
Aroclor 1248	0.4	0.5	Chick hatchability	Chapman, 2003
Aroclor 1254	0.6	1.2	Chick hatchability	Chapman, 2003

Note: Bolded values indicate the selected TRV.

[a]These values are interpreted as the interpolated dose resulting in a 10% or 25% decrease in endpoint response relative to the control group for the NOAEL and LOAEL, respectively; see Chapman, 2003.

[b]Chapman (2003) reports two dose-response patterns in the chicken studies for Aroclor 1242; this may be due to the difference in the batch tested, organisms, feed characteristics, or experimental design. Selected values are derived from the more sensitive response data.

Table 6-9 summarizes the selected mammalian TRVs for PCBs that were derived as part of the Chapman (2003) analysis; no mink study for Aroclor 1248 was identified but the author concluded that this mixture is as toxic as Aroclor 1254 based on *in vitro* bioassay data. The interpolated no effect and low effect doses for Aroclor 1254 (NOAEL – 0.080 μg PCB/g-day; LOAEL – 0.096 μg PCB/g-day) were selected as the mammalian TRVs for total PCBs.

Table 6-9. Summary of Mammalian TRVs for PCB Mixtures Based on Mink Data.

Aroclor Mixture	No Effect[a,b] (μg/g-d)	Low Effect[a,b] (μg/g-d)	Effect	Reference
Aroclor 1242	0.208	0.224	Decrease in live kit production	Chapman, 2003
Aroclor 1254	**0.080**	**0.096**	Decrease in number of live kits per mated female; kit birth weight	Chapman, 2003

Note: Bolded values indicate the selected TRV.

a. These values are interpreted as the interpolated dose resulting in a 10% or 25% decrease in endpoint response relative to the control group for the NOAEL and LOAEL, respectively; see Chapman, 2003.

b. Data converted from diet-based TRV to dose assuming that a female mink consumes 0.16 g/g body weight-day (average farm-raised individuals in Michigan [Bleavins and Aulerich, 1981]).

2,3,7,8-TCDD (herein referred to as TCDD) belongs to a class of compounds known as chlorinated dibenzodioxins that are ubiquitous in the environment as a result of various industrial processes (*e.g.,* solid waste incineration; the production, use, and disposal of pesticides and PCBs; during the bleaching process for paper manufacturing; and the production and recycling of metals). Dioxins are usually

generated concurrently with other chemicals known as chlorinated dibenzofurans, both of which are highly persistent and have been detected in all environmental media (*i.e.*, air, water, soil, animal tissue).

Laboratory toxicity data show that fish are generally more sensitive to TCDD than plants, aquatic invertebrates, and other aquatic vertebrates (*e.g.,* amphibians) (USEPA, 1993a). The high lipid content in fish makes them highly susceptible to bioaccumulation of TCDD in their tissues, which can essentially be transferred up the food chain to higher-trophic level organisms, such as birds and mammals (including humans). Effects of TCDD exposure to mammals and birds are similar to fish and include delayed mortality, a "wasting" syndrome characterized by reduced food intake and reduced body weight, reproductive toxicity, histopathological alterations, developmental abnormalities, and immunosupression (USEPA, 1993a).

Several long-term feeding studies have been conducted using a variety of bird species; information on the two most relevant studies with chickens and Ring-Necked pheasants is summarized in Table 6-10. The Nosek *et al.* (1992a,b) study was selected as the basis for establishing a TCDD TEQ TRV for birds.

A 10-fold subchronic to chronic extrapolation factor was applied to the pheasant NOAEL/LOAELs because the exposure duration was likely inadequate to achieve steady state conditions in the laying hens (USEPA, 1993a); moreover, the dose-response function appears to be very steep. The recommended TRVs (NOAEL – 1.4 x 10^{-6} µg TCDD/g-day; LOAEL – 1.4 x 10^{-5} µg TCDD/g-day) are consistent with the early life stage study with chickens exposed to 2,3,7,8- TCDF conducted by McKinney *et al.* (1976). Table 6-10 also summarizes the most relevant chronic feedings studies with mammals available for TCDD. A feeding study by Tillet *et al.* (1996) with mink is the basis for the recommended mammalian wildlife TRV for TCDD (NOAEL – 0.8 x 10^{-7}µg TCDD/g-day; LOAEL – 2.24 x 10^{-6}µg TCDD/g-day) because the mink is a receptor of concern at the site and the species is known to be sensitive to dioxin-like compounds.

Table 6-10. Summary of Chronic Feeding Studies with TCDD/TCDF.

Species	NOAEL (µg/g-d)	LOAEL (µg/g-d)	Effect	Reference
Birds				
Ring-necked pheasant (*Phasianus colchicus*)	**1.4 x 10^{-6}[a]**	**1.4 x 10^{-5}[a]**	Significant reduction in egg production; 100% embryotoxicity	Nosek *et al.,* 1992a,b
Chicken (*Gallus domesticus*)	0.1 x 10^{-5}[b]	0.1 x 10^{-4}[b]	Survival of newly hatched chicks to 21 days	McKinney *et al.,* 1976
Mammals				
Rat (Sprague Dawley)	0.1 x 10^{-5}	0.1 x 10^{-4}	Decreases in fertility in F_1 and F_2 generations	Murray *et al.,* 1979
Mink (*Mustela vison*)	**0.8 x 10^{-7}**	**2.24 x 10^{-6}**	Reduced kit body weights (3 wks) and reduced 9 survival (3 & 6 wks)	Tillet *et al.,* 1996

Note: Bolded values indicate the selected TRV.

a. Reported doses were based on exposures via interperitoneal injection and converted to an ingestion dose (USEPA, 1993a).

b. Based on dietary exposures to tetrachlorodibenzofuran (TCDF).

Table 6-11 presents a summary of the available LD_{50} studies results for various mammal species. The guinea pig appears to be most sensitive mammal (USEPA, 1993a), with the mink appearing to be only slightly less sensitive to the acute effects of TCDD exposure.

Table 6-11. Summary of Estimated Mammalian LD_{50} Benchmarks for TCDD TEQs.

Species	LD_{50} (μg/g)	Reference
Guinea pig (*Cavia porcellus*)	0.0006 – 0.002	As cited in USEPA, 1993a
Mink (*Mustela vison*)	0.0042	Hochstein *et al.,* 1988
Rat (*Rattus norvegicus*)	0.022 – 0.045	As cited in USEPA, 1993a
Rabbit (*Oryctolagus cuniculus*)	0.115	As cited in USEPA, 1993a
Mouse (*Mus musculus*)	0.114 – 0.284	As cited in USEPA, 1993a
Hamster (various species)	1.157 – 5.0	As cited in USEPA, 1993a

Dichlorodiphenyltrichloroethane (DDT) and its primary metabolites (DDD and DDE) are manufactured organochloride pesticides (collectively referred to as DDx). DDT use in the United States was banned in 1972, but it was still manufactured for export until the mid-1980s. DDT is a broad-spectrum insecticide that was very popular due its effectiveness, long residual persistence, low acute mammalian toxicity, and low cost. DDT has been widely used to control insects on agricultural crops such as peanuts, soybeans, and cotton, as well as sprayed to decrease the incidence and spread of diseases such as malaria by controlling mosquitoes.

Upon introduction into the environment, DDT will enter soil, water, or air. DDT and its metabolites are strongly adsorbed onto particulates in water and settle into sediments where they become essentially immobile. DDT is highly toxic to aquatic life, including both invertebrates (crustaceans) and vertebrates (fish, birds). Furthermore, DDT and its analogues accumulate in lipid tissues of fish and other organisms, and subsequently bioconcentrate up the food chain.

The best known effect of DDT toxicity is impairment of nerve impulse conduction. Effects of DDT on the nervous system have been observed in animals and can vary from mildly altered sensations to tremors and convulsions. Death in animals following high exposure to DDT is usually caused by respiratory arrest. In addition to being a neurotoxicant, DDT is capable of inducing marked alterations on reproduction and development, which is attributed to hormone-altering actions of DDT isomers and/or its metabolites (ATSDR, 2002a). Egg-shelling thinning in upper-trophic level birds is believed to have resulted in population crashes in the1960s and 1970s. Due to the ban on the production and use of DDT in the U.S. and other parts of the world, exposures of wildlife have been declining since the early 1970s, as evidenced by marked decreases in the levels of DDT compounds in fish, shellfish, aquatic mammals, and birds (ATSDR, 2002a).

The well-publicized decline in wild raptor populations, including the bald eagle, during the 1950s and 1960s was attributed partly to reproductive impairment, particularly eggshell thinning. Egg production, fertility, and hatchability were largely unaffected in numerous studies in a variety of bird species. However, increased embryolethality, decreased egg size, delayed oviposition after mating, and increased testicular effects were observed with some regularity among experimental studies in birds. Several authors speculated that the effects were due to DDT-induced hormonal imbalances, and in fact, blood hormone levels (estrogen, luteinizing hormone) were altered in three of four studies in birds consuming either DDT or DDE in the diet (ATSDR, 2002a).

The most extensively studied species include the mallard duck (*Anas platyrhyncus*), Japanese quail (*Coturnix coturnix japonica*), domestic fowl, brown pelican (*Pelecanus occidentalis*), and ringed turtle dove (*Streptopelia risoria*). The most commonly reported endpoints were lethality, neurological, and reproductive endpoints. Of particular interest are those effects that were observed consistently across species and in spite of variability in exposure scenarios. The significant health effects most consistently reported were lethality (several taxa), hepatic (liver enzyme induction and liver damage in birds),

endocrine (estrogenic effects in several taxa, and reduced thyroid weight and altered thyroid activity in birds), neurological (tremors in several taxa), reproductive (oviposition delay and eggshell thinning in birds), and developmental (reduced chick survival in birds, testicular feminization) (ATSDR, 2002a). Table 6-12 presents the TRVs and various feeding studies with DDT. The Anderson *et al.* study was selected as the basis for develop the NOAEL and LOAEL TRVs for bird receptors as the brown pelican is believed to be one of the most sensitive piscivorous bird species to DDT. The long-term reproduction study conducted by Fitzhugh (1948) of Sprague-Dawley rats evaluated multi-generational toxicity and sensitive endpoints and was selected as the basis for establishing mammalian TRVs for this evaluation.

Table 6-12. Summary of Chronic Feeding Studies with DDT.

Species	NOAEL (µg/g-d)	LOAEL (µg/g-d)	Effect	Reference
Birds				
Brown pelican (*Pelecanus occidentalis*)	**0.003**	**0.03**	Reproductive	Anderson *et al.*, 1975
Mallard duck (*Anas platyrhynchos*)	-	1.5	Reproductive	USEPA, 1995
Pelican (*Pelecanus occidentalis*)	0.009	-	Reproductive	USEPA, 1995
Mammals				
Rat (Sprague Dawley)	**0.8**	**4**	Reproductive	Fitzhugh, 1948; as cited in Sample *et al.*, 1996

Note: Bolded values indicate the selected TRV.

Dieldrin and aldrin are structurally similar and aldrin readily converts to dieldrin once it enters the environment or is ingested or inhaled by organisms. These compounds are discussed together because both are COPECs for the LPRRP. Dieldrin is an organochloride pesticide, belonging to the cyclodiene group of pesticides, which also includes endrin, endosulfan, and aldrin. Dieldrin is no longer produced or used, but was once used extensively as an insecticide on crops such as corn and cotton, and to control termites. Aldrin is a more effective pesticide than dieldrin and therefore was more extensively used as a soil insecticide (ATSDR, 2002b).

Many species of aquatic invertebrates concentrate dieldrin from very low water concentrations, yielding high concentration factors. The bioconcentration of dieldrin in aquatic organisms is principally from the water rather than by ingestion of food. Aldrin and dieldrin are both highly toxic to aquatic crustaceans and fish. Effects on mammals include liver damage, central nervous system effects, and suppression of the immune system. Dieldrin and aldrin also disrupt the endocrine and reproductive systems (ATSDR, 2002a).

TRVs for dieldrin have been developed by USEPA (2005c) as part of the process of developing Ecological Soil Screening Levels (Eco-SSLs) for mammals and birds for 23 contaminants using a transparent, ecologically relevant, and comprehensive process. The TRVs are listed in Table 6-13 below.

Table 6-13. TRVs for Dieldrin from USEPA Eco-SSL Derivation[a].

COPEC	Test Species	Wildlife Receptors	Low TRV[b]	High TRV[b]	Reference
Dieldrin	Mallard duck (*Anas platyrhynchos*)	Avifauna	**0.071**	**3.8**	Nebeker *et al.,* 1992
Dieldrin	Rat (Sprague Dawley)	Mammals	**0.015**	**0.030**	Harr *et al.,* 1970

[a] Rerelease of the Eco-SSL document is currently pending

[b] High TRVs are equivalent to the LOAEL from the study that the low TRV (NOAEL) was selected; units in µg COPEC/g body weight-day.

6.3 Risk Characterization

The risk characterization combines the exposure assessment with the toxicity assessment to derive a quantitative estimate of risk. Risks were derived based on both the high and low estimates of toxicity, to provide a NOAEL and LOAEL estimate of risk. Individual risk estimates to a given receptor for each chemical and for each exposure medium were calculated and then summed to provide a total cumulative estimate of risk, the hazard index.

6.3.1 Benthic Invertebrate Risk Estimates

Risks to benthic invertebrates were evaluated based on sediment benchmarks developed for marine and estuarine ecosystems (Table 6-14). For macroinverebrates, such as Blue crab, grass shrimp, and Eastern oyster, risks were based on estimates of critical body residues (CBRs) (Table 6-15). In general, a CBR is a contaminant- and taxon-specific threshold concentration measured in biological tissue above which adverse effects of ecological relevance would be anticipated to occur. This residue-based approach to evaluating risk provides a number of distinct advantages over the exposure-based approach, such as the explicit consideration of contaminant bioavailability and metabolism (McCarty and MacKay, 1993). The CBRs selected for this evaluation were based on conservative CBRs previously identified for the CSM Technical Memorandum (Battelle, 2006b) and are presented in Table 6-15.

Table 6-14. Summary of Hazard Quotients for Benthic Invertebrates

Chemical Parameter	*Marine/ Estuarine Values*		Lowest Sediment Benchmark[c] (µg/g)	Sediment EPC[d] (µg/g)	Hazard Quotient[e]	Relative Magnitude[f]
	NOAA ER-L[a] (µg/g)	NJ DEP[b] (µg/g)				
Copper	34	34	34	236	6.9	0.4%
Lead	47	47	47	375	8.0	0.4%
Mercury	0.15	0.15	0.15	3.6	24	1.3%
Low Molecular Weight PAHs	0.55	-	0.55	41	74	3.9%
High Molecular Weight PAHs	1.7	-	1.7	61	36	1.9%
Total PCBs	0.023	0.023	0.023	1.8	79	4.2%
Dieldrin	0.000020	-	0.000020	0.019	936	49.3%
Total DDx	0.0016	0.0016	0.0016	0.38	239	12.6%
TCDD TEQ (D/F)	0.0000032[g]	-	0.0000032	0.0016	493	26.0%
TCDD TEQ (PCBs)	0.0000032[g]	-	0.0000032	0.0000038	1.2	0.1%
TCDD TEQ	0.0000032[g]	-	0.0000032	0.0016[h]	494	

a. ER-L = Effects Range-Low from Long *et al.*, 1995.
b. NJ DEP Guidance For Sediment Quality Evaluations, November 1998. References Long et al, 1995.
c. Minimum of the ER-L and the NJ sediment benchmark values.
d. Exposure Point Concentration (EPC) is based on the 95% Upper Confidence Level on the arithmetic mean of the values in the assessment data set as discussed in the text. TEQs calculated using fish TEFs.
e. Hazard Quotient (HQ) is the ratio of the EPC to the benchmark value.
f. Percentage of the COPEC HQ to the sum of all Hazard Quotients (excluding the TCDD TEQ value).
g. Derived by USFWS using sediment chemistry for Newark Bay and oyster effect data presented in Wintermyer and Cooper, 2003.
h. TCDD TEQ for dioxin is based on a fish TEFs

Table 6-15. Summary of Critical Body Residues for Fish and Benthos Receptors.

COPEC	CBR (µg/g)		Species	Endpoint
	NOAEL	LOAEL		
American Eel/White Perch				
Copper	0.002	0.02	Channel catfish	Growth - LOED
Lead	0.028	0.28	Rainbow trout	Growth - ED_{11}
Mercury	0.006	0.06	Channel catfish	Mortality - LD_{50}
Methyl mercury	0.001	0.01	Mummichog	Growth – ED_{146}
Low Molecular Weight PAHs	0.21	2.1	Pacific Sand sole	Mortality - LD_{51}
High Molecular Weight PAHs	0.21	2.1	Pacific Sand sole	Mortality - LD_{51}
Total PCBs	0.0025	0.025	Japanese medaka	Reproduction – ED_{11}
Dieldrin	0.011	0.11	Rainbow trout	Growth - LOED
Total DDx	0.000039	0.0018	Japanese medaka	Mortality - LOED
TCDD TEQ – fish[a]	0.000034	0.000058	Lake Trout	Growth - LOED

Table 6–15. Summary of Critical Body Residues for Fish and Benthos Receptors, continued.

COPEC	CBR (µg/g)		Species	Endpoint
Mummichog				
Copper	0.002	0.02	Channel catfish	Growth - LOED
Lead	0.028	0.28	Rainbow trout	Growth - ED_{11}
Mercury	0.006	0.06	Channel catfish	Mortality - LD50
Methyl mercury	0.001	0.01	Mummichog	Growth – ED_{-146}
Low Molecular Weight PAHs	0.21	2.1	Pacific Sand sole	Mortality - LD_{51}
High Molecular Weight PAHs	0.21	2.1	Pacific Sand sole	Mortality - LD_{51}
Total DDx	0.16	0.85	Mummichog	Reproduction - ED_{20}
Total PCBs	0.0044	0.044	Fundulus	Reproduction – ED_{114}
Dieldrin	12.8	34	Sheepshead minnow	Mortality - LOED
TCDD TEQ – fish[a]	0.0000635	0.000635	Mummichog	Mortality - LOED
Benthic Macroinvertebrates				
Copper	0.086	0.86	Littleneck clam	Mortality - LD_{11}
Lead	0.52	5.2	Freshwater amphipod	Mortality - LD_{25}
Mercury	0.0095	0.095	estuarine copepod	Reproduction – ED_{50}
Methyl mercury	0.0095	0.095	estuarine copepod	Reproduction – ED_{50}
Low Molecular Weight PAHs	0.022	0.22	Blue mussel	Reproduction - LOED
High Molecular Weight PAHs	0.022	0.22	Blue mussel	Reproduction - LOED
Total DDx	0.00018	0.0018	Freshwater amphipod	Mortality – LD_{50}
Total PCBs	0.42	1.1	Grass shrimp	Mortality - LOED
Dieldrin	0.01	0.08	Pink shrimp	Mortality - LOED
TCDD TEQ - fish[a]	0.00000015	0.0000013	Eastern oyster	Reproduction - LOED

[a] Benchmark used to evaluate hazards posed by exposure to dioxin, furan, and coplanar congeners (*i.e.*, TCDD TEQ (D/F) and (PCBs).

Based on the magnitude of exceedance of the sediment benchmarks, dieldrin had the highest relative contribution of total risk (49.3%) with an HQ of 936. TCDD TEQ for dioxins and furans was the next largest contributor to the total risk comprising 26.0% of the over all risk. Copper and lead contributed the least with HQs of 6.9 and 8.0, respectively.

Current condition risk evaluated for the macroinvertebrates based on CBRs compared measure tissue concentrations to NOAEL and LOAEL body residues concentrations that are associated with adverse responses in morality, growth, and reproduction. The details of these analyses are provided in Attachment E and summarized in Figure 6-1 through Figure 6-3. Both the LOAEL and NOAEL estimates of risk were calculated, the total HI is 5,100 for the NOAEL scenario and 540 for the LOAEL. Total DDx and TCDD TEQ for dioxins and furans contribute the most to the LOAEL and NOEL HI; total DDx accounts for over 50% of the total HI and the TCDD TEQ accounts for approximately 30%. PAHs contributed the least with the LOAEL HI just above 1 for total PAHs.

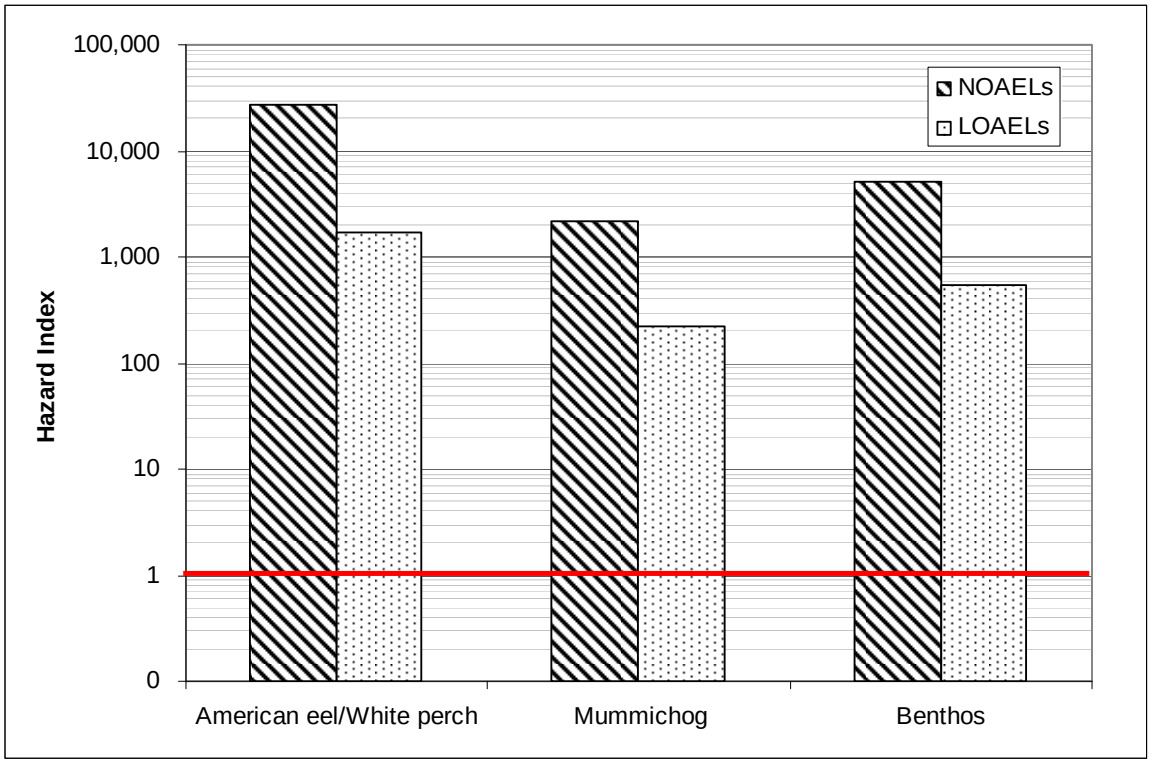


Figure 6-1. Hazard Indices for American Eel/White Perch, Mummichog, and Benthic Receptors Based on Tissue Residue Under Current Conditions.

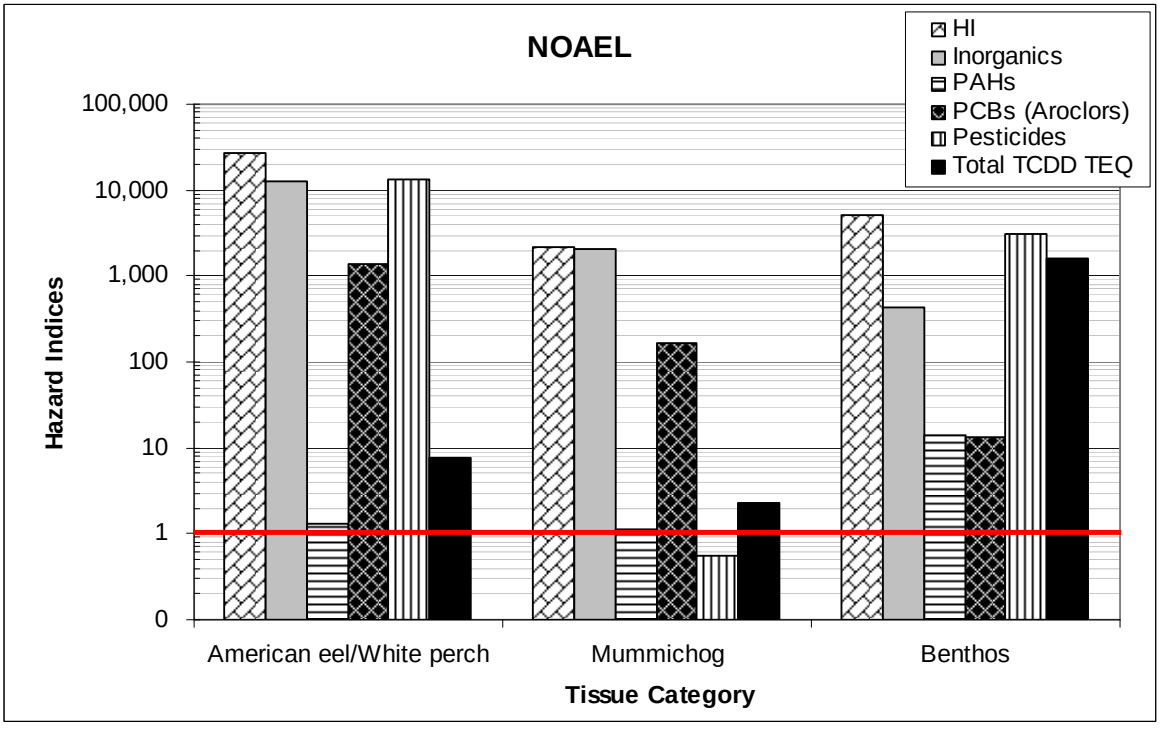


Figure 6-2. Hazard Ratios for American Eel/White Perch, Mummichog, and Benthic Receptors Based on NOAEL Tissue Residue Under Current Conditions

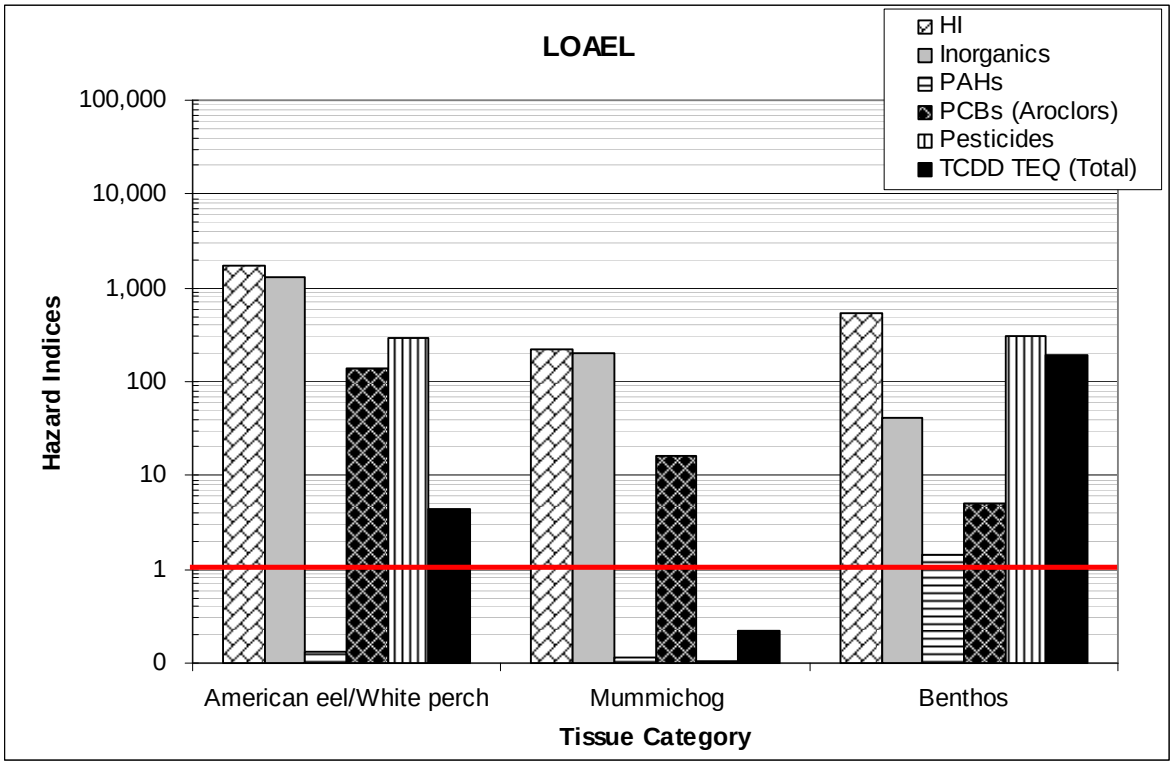


Figure 6-3. Hazard Ratios for American Eel/White Perch, Mummichog, and Benthic Receptors Based on LOAEL Tissue Residue Under Current Conditions

6.3.2 Fish Risk Estimates

Risks evaluated for forage fish and for the large AE/WP fish receptor are based on estimates of critical body residues (CBRs) (Figure 6-1 through Figure 6-3). As discussed in the previous section for benthic invertebrates, both LOAEL and NOAEL estimates of risk were calculated for the two fish receptors, based on CBR data. Results are presented in detail in Attachment E.

For the mummichog under current conditions, the total HI is 2,200 for the NOAEL scenario and 220 for the LOAEL. Copper contributes the most to the NOAEL and LOAEL (approximately 88% for both). PCBs contribute the next largest portion to the total risk (7%) whereas the pesticides (total DDx and dieldrin) and the PAHs have an HQ of 1.1 or less. For the AE/WP receptor, the total HI is 28,000 for the NOAEL and 1,700 for the LOAEL. Copper and total DDx account for over 90% of the total risk for both the LOAEL and NOAEL scenarios.

6.3.3 Wildlife Risk Estimates

Current risks calculated for the mink and the Great Blue heron (Attachment F) are summarized in Figure 6-4 through 6-6. For the mink, the total HI across all chemicals and exposure scenarios is 1600 for the NOAEL and 72 for the LOAEL. For both the LOAEL and NOAEL exposures, the majority of risks are associated with total TCDD TEQ (80% and 99%, respectively), with dioxin/furan compounds accounting for over 50% of the TEQ in both cases. Total PCBs comprise 17% of the LOAEL risk and 1% of the NOAEL risk. For the LOAEL and NOAEL risks, the other COPECs (copper, mercury, lead, dieldrin, HPAH, LPAH, total DDx) have a combined hazard quotients slightly above 1.0.

The fish consumption pathway contributes to the majority of the risks to the mink, accounting for 61% and 63% of the total risk for the NOAEL and LOAEL scenarios, respectively. Minimal risk is associated with sediment exposures (8%), with HQs below 1 for all COPECs except for total TCDD TEQ.

Two scenarios were evaluated for the great blue heron: the first was based on a diet comprised primarily of mummichogs, and the other is based on an AE/WP fish diet (Figure 6-4 through Figure 6-6). For the AE/WP fish diet, the total risk is 150 for the NOAEL and 16 for the LOAEL. TCDD TEQ (PCBs) is the primary risk driver, contributing more than 55% each for the NOAEL and LOAEL risks. For both the NOAEL and LOAEL, TCDD TEQ (for dioxin/furan) contribute 18% and 17%, respectively, to the total risk. For the NOAEL, the HQ for total DDx (HQ=20), mercury (HQ=6.5), total PCBs (HQ=3.9), and lead (HQ=1.2) were all above 1.0. For the LOAEL, only the HQs TCDD TEQ for PCB and dioxin/furan and total DDx were greater than 1.0. The remaining compounds (mercury, lead, cooper, dieldrin, LPAHs, and HPAHs) had HQs less than 1.0. The fish consumption pathway contributes to the majority of the risk (> 60%), and HQs associated with sediment exposures are below 1.0 for all COPECs except the NOAEL TCDD TEQs (both D/F and PCBs), mercury, and lead which are both slightly above 1.0.

Assuming that the great blue heron consumes primarily mummichogs, the risks are lower, with a total HI of 78 for the NOAEL and 8.6 for the LOAEL, respectively. As with the AE/WP fish diet, TCDD TEQ (PCBs) is the primary risk driver for the mummichog diet, contributing to 59% of the total NOAEL risk and 53% of the LOAEL risk. TCDD TEQ (D/F) contributes 24% to the NOAEL risk and 22% to the LOAEL risk. For the NOAEL, there is an added risk from lead, mercury, and TCDD TEQ (PCBs) with HQs above 1.0. For the LOAEL, all COPECs the HQs are below 1.0.

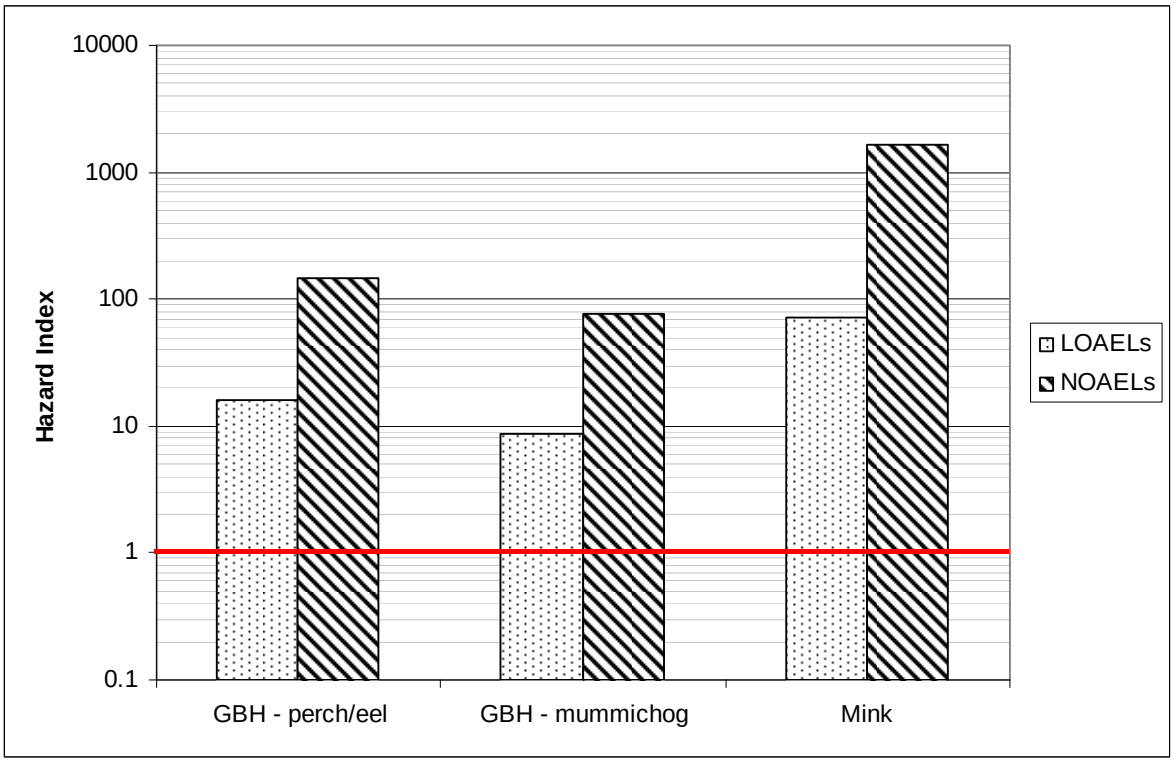


Figure 6-4 Hazard Indices for Wildlife Receptors Under Current Conditions.

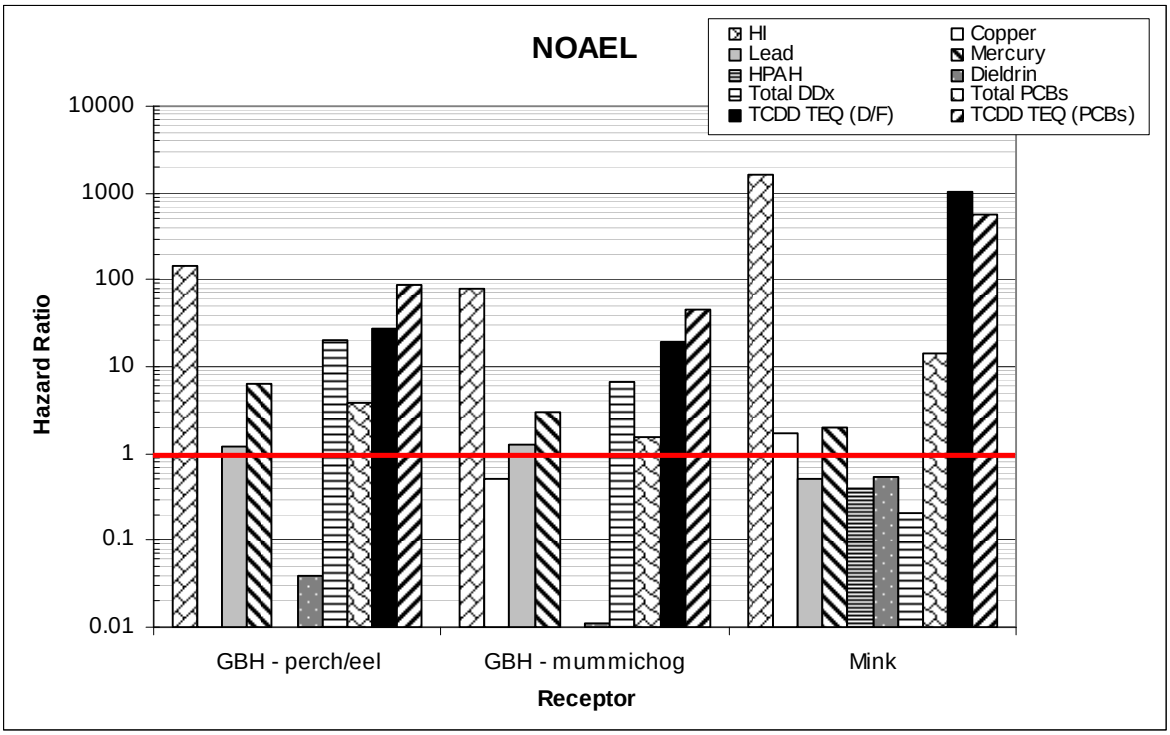


Figure 6-5. Hazard Ratios for Wildlife Receptors Based on NOAEL Tissue Residue Under Current Conditions.

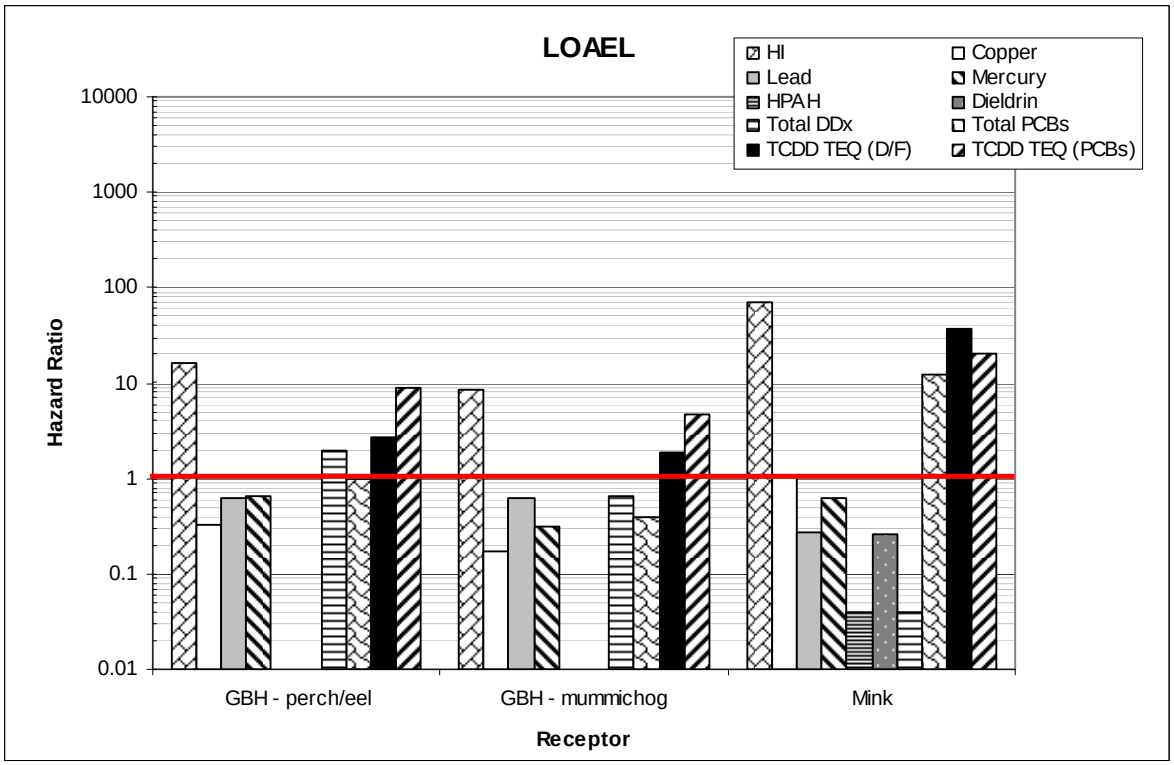


Figure 6-6. Hazard Ratios for Wildlife Receptors Based on LOAEL Tissue Residue Under Current Conditions.

6.3.4 Summary of Current Risks

The current risks for ecological receptors are summarized in Table 6-16 for the benthic invertebrates, AE/WP, and mummichog receptors and the details are presented in Attachment E. Table 6-17 provides a summary of the current risk to wildlife receptors. The details are presented in Attachment F. Under current conditions, risks for the benthic invertebrates, AE/WP, and mummichogs are driven by copper and total DDx. For the wildlife receptors the risks are primarily driven by TCDD TEQ (D/F) and TCDD TEQ (PCBs). Risks to mink are greater than those for the heron and are primarily associated with TCDD TEQ (D/F), whereas TCDD TEQ (for PCBs) are the primary risk drivers for the heron.

Table 6-16. Summary of Ecological Risk Estimates for Benthic Invertebrates, AE/WP, and Mummichog Receptors Under Current Conditions.

	Benthic Invertebrates			American Eel/White Perch (AE/WP) Receptor		Mummichog Receptor	
	Sediment Benchmarks	Macroinvertbrates					
COPECs	HQ	NOAEL HQ	LOAEL HQ	NOAEL HQ	LOAEL HQ	NOAEL HQ	LOAEL HQ
Inorganic Compounds							
Copper	6.9	410	41	12400	**1200**	**1900**	**190**
Lead	8	1.0	0.1	23	2.3	45	4.5
Mercury	24	10	1.0	350	35	41	4.1
Semivolatile Organic Compounds (PAHs)							
Low molecular weight PAHs	74	6.9	0.69	0.82	0.082	0.82	0.082
High molecular weight PAHs	36	74	0.74	0.48	0.048	0.31	0.031
Polychlorinated biphenyl (PCB)							
Total PCBs (sum Aroclors)	79	13	5	1400	140	160	16
Pesticides/Herbicides							
Dieldrin	936	2.2	0.28	2.5	0.25	0.00033	0.00012
Total DDx	239	**3000**	**300**	**13000**	290	0.55	0.1
Dioxin-Like Compounds							
TCDD TEQ (D/F)	493	1500	170	7.4	4.3	2.2	0.22
TCDD TEQ (PCBs)	1.2	170	19	0.15	0.088	0.027	0.0027
Total HI	**1,897**	**5,187**	**538**	**27,184**	**1,672**	**2,150**	**215**

Bolded cells indicate the most significant contribution towards total risk for the receptor.

Table 6-17. Summary of Ecological Risk Estimates Wildlife Receptors Under Current Conditions.

COPECs	Mink Recptor		Great Blue Heron American Eel/White Perch (AE/WP) Diet		Great Blue Heron Mummichog Diet	
	NOAEL HQ	LOAEL HQ	NOAEL HQ	LOAEL HQ	NOAEL HQ	LOAEL HQ
Inorganic Compounds						
Copper	1.7	1	0.97	0.32	0.52	0.17
Lead	0.52	0.27	1.2	0.61	1.6	0.63
Mercury	2	0.62	6.5	0.65	3.1	0.31
Semivolatile Organic Compounds (PAHs)						
Low molecular weight PAHs	--	--	--	--	--	--
High molecular weight PAHs	0.04	0.04	--	--	--	--
Polychlorinated biphenyl (PCB)						
Total PCBs (sum Aroclors)	15	12	3.9	0.98	1.6	0.39
Pesticides/Herbicides						
Dieldrin	0.53	0.26	0.039	0.00074	0.011	0.00021
Total DDx	0.2	0.04	20	2	6.5	0.65
Dioxin-Like Compounds						
TCDD TEQ (D/F)	**1000**	**37**	27	2.7	19	1.9
TCDD TEQ (PCBs)	560	20	**87**	**8.7**	**46**	**4.6**
Total HI	**1,580**	**71**	**147**	**16**	**78**	**9**

6.4 Ecological Uncertainty Analysis

This section discusses limitations of the analyses, describes the primary sources of uncertainties, and evaluates whether these uncertainties and limitations may have resulted in an over- or underestimation of risk. Uncertainties in the quantification of risk associated with the analysis are identified and their impacts on risk estimates are discussed below.

Uncertainties associated with the problem formulation (including development of the conceptual site model, receptor identification, and the selection of COPECs, exposure assessment, effects assessment, and overall risk characterizations are discussed. Table 6-18 summarizes the principal ecological risk uncertainties and identifies the projected impact on the ecological risk conclusions.

As with the HHRE, a significant uncertainty associated with the ecological evaluation is the decision to focus the analysis on a limited subset of COPECs. As a result, the evaluation did not attempt to quantify total site risk (as per the Comprehensive Environmental Response, Compensation, and Liability Act [CERCLA]) but rather to determine whether existing (current) conditions pose sufficient hazard to warrant consideration of a remedial action. In addition to focusing on a subset of COPECs, the analysis also did not evaluate all potentially complete exposure pathways (*e.g.,* surface water) or ecological receptor categories or life stages (*e.g.,* early life stage exposures). Although conservative assumptions were employed throughout the evaluation, the limited focus of the analysis indicates that there is a low to

moderate level of uncertainty and that, overall, the risk evaluation tended to underestimate ecological hazards associated with these elements (Table 6-18).

Several parameters associated with the exposure assessment have uncertainties associated with them that impart uncertainty to the calculated risks, including EPCs, potential receptors, and exposure assumptions evaluated in the risk assessment. Each of these is discussed below and summarized on Table 6-18.

- Based on USEPA risk assessment guidance, the UCL of the arithmetic mean is used as the EPC because it is a conservative estimate of the average site-wide concentration that a receptor would be exposed to. As discussed for the HHRE, the amount of uncertainty in the calculated risks resulting from uncertainty in the EPCs is considered low.
- Risk estimates for individual mink which only consume white perch would be underestimated because concentrations of COPECs in white perch were always higher than in the American eel. Averaging the two fish species would therefore dilute the EPC. On the other hand, the risk for those individuals consuming only American eel would be overestimated. Exposures would also be overestimated to the extent that wildlife receptors consumed more migratory species such as striped bass which tend to have lower tissue COPEC concentrations.
- GBH exposure scenario which assumes site fidelity of 100% (SUF=1) which may lead to overestimates of risk because UCL levels in river are assumed to be higher than other regional sources of food GBH could encounter.

As discussed below and summarized on Table 6-18, the primary aspects of the toxicity assessment that impart uncertainty to the calculated risks include uncertainty in the toxicity data for constituents detected at the site.

- TRVs are typically based on results of tests performed on test animals and extrapolated to wildlife species; selected values are generally conservatively developed as the lowest LOAEL for well-conducted studies that evaluated ecologically-relevant endpoints. Because the most conservative values available are typically used, risks are more likely to be overestimated than underestimated. In the case of the mink receptor, well-conducted toxicity test results are available and were used to develop the TRVs.
- In July 2006, the WHO released their re-evaluation of human and mammalian TEFs for dioxins and dioxin-like compounds performed in 2005. The risk evaluation was completed using the 1998 TEFs. An analysis of the potential impacts of these revised TEFs would have on the ecological risk evaluation is provided in the Human Health Uncertainty section of this report. It is concluded that the new TEFs would have no substantive change on the outcome of this evaluation.
- Use of the most sensitive species to select CBRs likely resulted in the residue-based analysis overestimating risks. Species such as salmon and trout are not found in the Lower Passaic River, and hazards identified in the residue-based analysis for the AE/WP are likely overestimated. A separate set of CBRs was also developed for estuarine forage fish such as *Fundulus* spp. and CBRs for these species were, in some cases, higher than those for the AE/WP (such as for TCDD and total DDx).
- In several cases, CBR NOAELs were estimated using an assumed 10-fold extrapolation factor; this may have under- or overestimated hazards in the evaluation. In addition, the literature studies queried in the tissue residue effects databases vary in terms of quality and relevance to the study objectives. Although the conservative procedures employed in the selection of CBRs tended to result in risks being overestimated, suitable tissue residue data for certain COPECs were limited and may not have included relevant sensitive species or life stages.

Finally, uncertainty in the calculated risks can arise from uncertainty in the way in which risks are calculated or aggregated, as discussed below and on Table 6-18.

- A portion of the calculated risks may be attributed to the presence of naturally occurring constituents or constituents that are present at the site because of regional anthropogenic sources (*e.g.,* mercury). The effect of including background and ambient constituents in the risk evaluation is that the calculated risks overestimate the site-related risks that are due to chemical releases. The significance of this effect is explored more fully in the residual risk analysis in Section 8.0.

Table 6-18. Summary of Major Uncertainties in the Ecological Risk Evaluation and Estimated Impacts on Calculated Risks.

Risk Evaluation Step	Source of Parameter Uncertainty	Description of Uncertainty	Impact on Calculated Risks
Problem Formulation	Identification of COPECs for quantitative evaluation	Only a subset of contaminants likely comprising the primary risk drivers at the site were selected and evaluated.	Risks are somewhat underestimated; however exposures to the selected COPECs likely represent a substantial majority of the total hazards posed to ecological receptors.
		COPECs associated with other environmental media (*e.g.,* surface water) were not considered.	Risks are underestimated.
	Mercury and methyl mercury	Due to lack of methyl mercury data in the biota tissue data, results for mercury were used as surrogate methyl mercury. This assumes that all mercury bioaccumulated in the food chain is present as methyl mercury.	Although the hazards may be overestimated, the overall uncertainty is considered low because methyl mercury generally constitutes a substantial majority of the mercury bioaccumulated in fish tissue.
	Evaluated exposure pathways	Other potentially complete exposure pathways for fish and wildlife ad fish were not included (*e.g.,* dermal contact with sediment; consumption of contaminated drinking water). In addition, exposure to dioxin and dioxin-like compounds in sensitive critical life stages (*e.g.,* fish embryos) was not explicitly evaluated.	Exclusion of these additional pathways would underestimate the risks for the site.
	Receptors and life stage evaluated	Wildlife species with foraging habits other than piscivorous were not evaluated.	It is anticipated that wildlife consumption of aquatic prey including fish and shellfish would result in the highest dietary exposures to COPECs and it is likely that risk to other wildlife species are of lower magnitude than reported in this evaluation.
Exposure Assessment	EPCs for biota tissue	95% UCLs were calculated from measured data collected from numerous samples distributed across the exposure area and used as the EPC to calculate risk.	Risks for some compounds with low frequency of detection may be over- or underestimated because it was assumed that samples reported as "ND" contained a concentration equal to one-half the detection limit.

Table 6-18. Summary of Major Uncertainties in the Ecological Risk Evaluation and Estimated Impacts on Calculated Risks, continued.

Risk Evaluation Step	Source of Parameter Uncertainty	Description of Uncertainty	Impact on Calculated Risks
	Use of a AE/WP fish composite	Use of EPCs based on a combination of white perch and American eel tissue data to represent exposures to piscivorous wildlife assumes that from the Lower Passaic River and that each of these species is equally consumed.	Risk estimates for individual mink which only consume white perch would be underestimated because concentrations in white perch were always higher than the American eel. Averaging the two fish species would therefore dilute the EPCs. On the other hand, the risk for those individuals consuming only American eel would be overestimated. Exposures would also be overestimated to the extent that wildlife receptors consumed more migratory species such as striped bass which tend to have lower tissue COPEC concentrations.
	Receptors exposure parameters	Selecting the most representative exposure parameters for the angling activities/habits is difficult, especially for exposure duration, exposure frequency, and fish ingestion rates.	Risk estimates were based on conservative values derived from standard ecological risk guidance (USEPA, 1993b) or professional judgment. It is likely that hazards were overestimated by the general tendency to select conservative values.
	Use of historical data	Sediment samples dating back to 1994 and biota tissue samples dating back to 1995 were used to develop EPCs in the evaluation. These data are up to 12 years old and may not be representative of current conditions.	Inclusion of the historical data may tend to over-estimate current exposures and hazards based on trends observed in sediment cores. Calculated multipliers to translate 1995 sediment concentrations to equivalent present-day concentrations range from 0.6 (total PCBs) to 1.0 (DDT); the estimated average multiplier for TCDD is 0.9. The use of historical data would have different impact on the calculated risks depending on which COPECs were identified as the primary risk drivers.
	Wildlife diet composition	Literature was referenced to quantify the relative proportion of fish and shellfish in the diets of the modeled wildlife receptors.	Range of estimated values generally did not differ dramatically (ranging from 0 to 30% in different studies depending on the particular habitat) and the tissue EPCs are fairly comparable. However, this uncertainty has more significance for the future residual risk analysis because of significant differences in the estimated bioaccumulation factors (BAFs) for higher trophic level fish and shellfish. This uncertainty is discussed further in Section 7.0.

Table 6-18. Summary of Major Uncertainties in the Ecological Risk Evaluation and Estimated Impacts on Calculated Risks, continued.

Risk Evaluation Step	Source of Parameter Uncertainty	Description of Uncertainty	Impact on Calculated Risks
	Fish prey trophic level	Wading birds generally take smaller forage fish rather than larger higher trophic status species. Concentrations in mummichog (a forage fish) are approximately an order of magnitude lower than in white perch and American eel	Use of the fish EPCs based on higher trophic level dataset likely overestimates risks to wading birds such as the heron. The magnitude of this impact was evaluated by also including an assessment of a diet that consisted of mummichogs.
Toxicity Assessment	Ingestion toxicity data	TRVs are typically based on results of tests performed on test animals and extrapolated to wildlife species; selected values are generally conservatively developed as the lowest LOAEL for well-conducted studies that evaluated ecologically-relevant endpoints.	Because the most conservative values available are typically used, risks are more likely to be overestimated than underestimated. In the case of the mink receptor, well conducted toxicity test results are available and were used to develop the TRVs.
	1998 vs. 2005 TEF values	The WHO released their re-evaluation of human and mammalian TEFs for dioxins and dioxin-like compounds performed in 2005.	An evaluation of the hazards posed based on use of the 2005 TEF values demonstrate that they are comparable to those based on the 1998 values.
	CBR effect thresholds	CBRs were selected based on a review of several large compilations of tissue residue effect data. Study quality is variable and relevance of particular endpoints uneven relative to the assessment endpoints.	Likely risks were overestimated; however, suitable tissue residue data for certain COPECs were limited and may not have included relevant sensitive species or life stages.
		Use of toxicologically unbounded study results to develop CBRs.	In several cases, NOAELs were estimated using an assumed 10-fold extrapolation factor; this may have under- or overestimated hazards in the evaluation.
		In general the most sensitive saltwater or estuarine fish species was selected to develop the CBRs. In many cases, CBRs are based on exposure to salmonid species that are known to be sensitive to COPECs such as dioxins, DDT, and mercury.	Species such as salmon and trout are not found in the Lower Passaic River and hazards identified in the residue-based analysis for the AE/WP are likely overestimated. A separate set of CBRs was also developed for estuarine forage fish such as *Fundulus* spp. and CBRs for these species were, in some cases, higher than for the AE/WP (such as those for TCDD and total DDx).

Table 6-18. Summary of Major Uncertainties in the Ecological Risk Evaluation and Estimated Impacts on Calculated Risks, continued.

Risk Evaluation Step	Source of Parameter Uncertainty	Description of Uncertainty	Impact on Calculated Risks
Risk Characterization	Distinguishing site-related risks from background and/or ambient risks	A portion of the estimated hazards may be attributed to the presence of naturally occurring constituents or constituents that are present at the site because of regional anthropogenic sources (*e.g.*, mercury).	The effect of including background and ambient constituents in the risk evaluation is that the calculated risks overestimate the Site-related risks that are due to chemical releases. The significance of this effect is explored more fully in the residual risk analysis.

7.0 DEVELOPMENT OF EPCs FOR FUTURE CONDITIONS

To assess risks to human health and ecological receptors following remedial actions, future sediment concentrations were derived and used to develop future EPCs. Future sediment concentrations were estimated by applying future cast multipliers to the current chemical concentrations (95% UCLs) to provide an estimate of the future chemical concentrations in sediment. Malcolm Pirnie, Inc. developed the future cast multipliers for 2,3,7,8-TCDD, total PCBs, total DDx, and methyl mercury. Two other COPECs, dieldrin and chlordane, did not have sufficient data to conduct extrapolations. Because the most robust and comprehensive dataset of COPEC concentrations in surface sediment was collected in 1995 (historical 1995 Tierra Solutions Inc. [TSI] dataset), a method to estimate the current concentrations was needed to calculate EPCs. The multipliers were developed to predict the 2005 chemical concentrations based on sediment chemistry data from 1995 and then applied to estimate sediment chemical concentrations in 2018, 2026, and 2035 for Target Area 1, Target Area 3, and the Fine-grained Sediment Areas.

THIS SECTION WILL BE COMPLETE ONCE THE RESULTS OF THE FUTURE CAST MODEL IS MADE AVAILABLE

8.0 REMEDIAL ALTERNATIVES FUTURE RISK EVALUATION

This section describes the results of a quantitative risk evaluation performed to assess the relative magnitude of risk currently associated with the Lower Passaic River compared to the relative reduction of risk associated with remediation of sediment within three target areas. The objective of the risk evaluation is to provide an assessment of the overall protection of human health and the environment considering a "no action" approach versus remediation of contaminated sediment present within three target areas to address requirements in NCP Section 300.430(e)(9)(iii). The remedial action objectives have been summarized in the text of the Draft FFS. Target areas also have been defined in the text of the Draft FFS and in Section 4.0. The results of the evaluation will be used to assist risk management decisions regarding the selection of a remedial action. Note that the goal of the alternative risk evaluation is not to provide an absolute estimate of risk reduction, but rather to provide order of magnitude estimates that incorporate considerable professional judgment and uncertainty.

THIS SECTION WILL BE COMPLETED ONCE THE RESULTS OF THE FUTURE CAST MODEL IS MADE AVAILABLE

9.0 CONCLUSIONS

Results of both the HHRE and the ERE support the conclusion that current conditions within the Lower Passaic River pose significant risks to human and ecological receptors. Although these evaluations did not attempt to quantify total site risks, it is evident that that magnitude of the current risks posed by the existing sediment inventory, and in particular, the bioaccumulation pathway associated with the fish and shellfish consumption pathway represents an exceedence of the risk range to both human health and the environment. The uncertainties associated with these calculations are also provided in the risk characterization.

THIS SECTION WILL BE UPDATED ONCE THE RESULTS OF THE FUTURE CAST MODEL IS MADE AVAILABLE

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